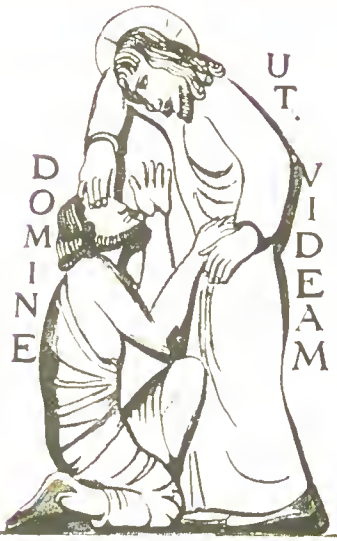
Subjective Symptoms

in

Eye Diseases.



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SUBJECTIVE SYMPTOMS IN EYE DISEASES:

Being Chapters on the Disorders of Vision Symptomatic of Diseases in the Eye and Central Nervous System.

BY

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PREFACE.

THESE chapters on Subjective Symptoms in Eye Diseases are to a great extent reprints, with but slight alterations, of papers which have appeared from time to time in the *Edinburgh Medical Journal*. I had originally intended to extend them, so as to render some of the more condensed and generalized sections more intelligible to those whose experience of ophthalmic medicine and surgery is limited to cases met with in the course of general practice. It was also my intention to have given to them at the same time a somewhat more exhaustive character than they at present possess. Other work prevents my carrying out these intentions at present, but I trust that, even in the fragmentary state in which the chapters now appear, they may be of interest to some whose professional reading is sufficiently extensive to enable them to devote some time to ophthalmological subjects.

EDINBURGH, *October* 1886.

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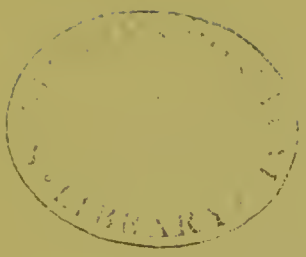
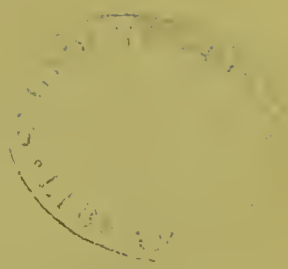



SUBJECTIVE SYMPTOMS IN EYE DISEASES.

INTRODUCTION.

SINCE the invention of the ophthalmoscope, the objective examination of every part of the eye has reached a degree of perfection which has no parallel for any other organ of the body. It is not unnatural to find, therefore, the attention of medical men nowadays less directed towards the subjective symptoms which accompany any pathological change in the eye than was the case formerly, when the observation of the cause lay more frequently beyond their power. Now that we are, however, able to detect an objective cause for many such subjective symptoms, they have become invested with a new interest, and their study, far from being superfluous, tends to throw light on many points connected, not only with the pathology, but also with the physiology of vision.

Nowhere has an attempt to make *much* out of ophthalmoscopic examination alone been more evident than amongst physicians, as an aid to the diagnosis and localization of disease elsewhere. There can be little doubt that the information to be gained in this respect by ophthalmoscopic examination alone has been much exaggerated. When we except optic neuritis, the retinitis occurring in Bright's disease, and the rare cases of tubercle of the choroid, in which position the tubercles probably never make their appearance until long after there has been good evidence of tuberculosis elsewhere, we have practically exhausted the list of cases in which the objective examination of the fundus may afford *per se* important aid in establishing or confirming a diagnosis. In

some cases retinal and choroidal changes may, it is true, point to a syphilitic origin of an obscure affection, but there are usually other signs to be found, which are at least quite as suggestive in this respect. I have excluded from the list atrophy of the optic nerve, because it is only in well-marked and advanced cases that we are justified in diagnosing atrophy without ascertaining what may be the state of functional activity, or, in other words, without a thorough subjective examination. It is not by any means rare to hear appearances pronounced as indicative of partial or incipient atrophy which a proper subjective examination would show to belong to the category of physiological variations. Many seem again to be under the impression that they can not only diagnose with tolerable certainty conditions of anæmia and hyperæmia of the fundus oculi, but that they are also at liberty to infer that these actual or assumed circulatory changes are conclusive evidence of similar conditions existing in the brain. Yet, notwithstanding the intimate connexion which exists between the vessels of the brain and the eyes, the sanguine expectations of many, and the assurances of a few, such as Bouchut, for whom cerebroscopy is synonymous with ophthalmoscopy, as to the possibility of diagnosing the existing vascular state in the brain from ophthalmoscopic examination of the fundus of the eye, have not been realized or confirmed. This is partly owing, no doubt, to the ordinary methods of ophthalmoscopic examination not giving a sufficiently magnified image to admit of the diagnosis of any but the most marked circulatory changes, but mainly to vascular changes in the brain being by no means always associated with similar changes in the vessels of the eye, and *vice versâ*. Some of the most important changes in the circulation of the brain, such as thrombosis of the sinuses, do in fact take place without revealing themselves by circulatory disturbances in the eye.

But whilst there is a tendency in some to exaggerate the importance of the information to be gained by an ophthalmoscopic examination, there are many who, from want of familiarity with the appearances of the fundus or other parts of the eye in health and disease, are inclined to place perhaps too little confidence in

their own objective examinations. A little more attention than is usually given to the various methods of examining accurately into the state of functional activity of the visual apparatus is to be advocated in both cases, as not only likely to lead to information interesting in itself, but also to be of value in directing them to the correct diagnosis of the cases which come under their treatment.

Besides, however, affording evidence of the existence or not of pathological conditions in the eye itself, abnormalities found by subjective examination in the state of the visual functions, may be of such a nature as to reveal not only the presence of, but point more or less definitely to the site of lesions in the brain, and this, too, in cases when absolutely no clue is given by an ophthalmoscopic examination alone.

A certain amount of intelligence on the part of the patient is necessary, of course, for a satisfactory subjective examination of any nature, and we must also be on our guard against unconscious or even wilful deception. We must remember, too, that in all such examinations we can only count upon obtaining approximations to the actual state of matters. We cannot expect, nor need we aim at the accuracy to be got from physical experiments. Thus, in an examination of the field of vision, for example, we ought not to infer that there is an existing limitation of peripheral vision in any direction, unless the extent of the field is found considerably less in that direction than normal, and is otherwise normal in extent. Still less are we justified in coming to a hasty assumption of a concentric limitation, or the existence of improvement on subsequent examination, as there is a great individual difference in the power of observation in tests of that nature, and a greater accuracy generally acquired by practice. For the rest, many objective methods are subject to the same sources of error. It is not an uncommon thing, for instance, to meet men who believe they are able to determine the state of refraction to 1 dioptre, or even to $\frac{1}{2}$ dioptre, and yet a little consideration of the optical conditions would show that this degree of approximation is one which it could only be possible to attain to in cases where

the ametropia did not exeeed a medium amount. Besides, there are sourees of error whieh render the result of a determination of this nature uneertain, so that even the most praetised observer will be found on trial to make mistakes, not only in estimating the higher, but also the lower degrees of ametropia. This belief in their own powers of determining objeetively the degree of ametropia leads some aetually to recommend the optieal eorrection inferred in this way, and that even when it is possible to make a simple subjeetive examination of the refraetion, a practiee whieh eannot be too strongly condemned. Another instanee in which an objeetive test is liable to lead to fallaeious results, which must therefore be reeeived with eaution, and all the more so, as unlike the objective examination of refraetion it is not eapable of verification by the applieation of subjeetive tests, is the estimation of the degree of eonvergenee of whieh a patient is eapable, or the strength of prism whieh he is able to overcome in the interests of binoeular vision. So mueh depends in these eases on the strength of will exerted to evolve momentarily the greatest amount of museular effort at the disposal of the individual. On this aeeount we must not be too ready to infer that an increased amount of convergence elieited by an examination undertaken, let us say, after any therapeutic interferenee is aseribable altogether to sueh interferenee, as, unless very marked indeed, it may depend only on the greater efforts made by the patient.

It has not, of eourse, been my intention in the foregoing remarks to underrate in any way the value of ophthalmoscopie or other objective methods of examination, but merely to insist on the importanee of not negleeting the subjeetive methods as well, as by so doing much important information may eseape deteetion. At the same time, while every endeavour should be made to attain to the greatest possible profieieney in objeetive examination, it is necessary to keep constantly before one the eirenmstanees which may vitiate to a greater or less extent the results of such examinations, so as not to place undue reliance in them.

CHAPTER I.

PAIN.

WHENEVER pain is complained of in the eye, it is only right that an examination should be made of its functions, with reference not only to its visual capabilities, central and peripheral, as well as qualitative and quantitative, but also to the dioptric, accommodative, and external muscular arrangements on which the normal performance of these functions greatly depends.

To those unaccustomed to make such examinations, and who, although able to use the ophthalmoscope, have not sufficient practice to justify them in placing complete confidence in the result of their own examinations of the fundus of the eye, there is often a feeling that the pain complained of by their patients is due to some deep-seated inflammation or other pathological process which they are unable to detect. The fact is, however, that pain in the eye, unaccompanied by appearances of external inflammation, or more or less sudden visual defects, is only very rarely the result of changes which require the use of the ophthalmoscope for their detection; and, indeed, as a general rule, always excluding the cases where there is a visible inflammation, the greater the pain complained of, the less likely is it to depend upon any pathological condition at all which can be referred to the eye.

In making an analysis of the causes of pain, we may distinguish two great groups: 1, non-inflammatory pain; 2, inflammatory pain.

In a large proportion of the cases of pain unaccompanied by inflammation, the pain only comes on or is at all severe when the eyes are used for work near at hand, such as reading or sewing. In other cases, again, the pain is independent of the use of the eyes. The name which is generally given to the non-inflammatory pain associated with the use of the eyes is *asthenopia*, which literally means a want of power in the eye to perform its functions.

We may distinguish three forms of asthenopia: 1, accommodative; 2, muscular; and, 3, nervous or retinal asthenopia.

Accommodative Asthenopia.—Asthenopia is generally inferred to be accommodative when the condition of the eyes is such as to necessitate and bring into action a stronger stimulus to accommodation than is required normally under similar conditions. Cases of hypermetropia and of paresis of accommodation come under this category. The latter, besides being much less common than the former, are not so often associated with asthenopia as might be expected, greatly because attempts at accommodation are not persisted in to the same extent as in hypermetropia. In hypermetropia the asthenopia is probably more frequently due to the maintaining of an abnormal relation between accommodation and convergence of the optic axes on the object fixed than to the keeping up of a larger proportion than normal of the available accommodative power, though probably to some extent to both these circumstances combined. That such is the case is shown by the fact, that when a hypermetrope squints he is freed from asthenopia, although making use of as much accommodation as others with the same degree of refractive error, who do not squint. The two impulses, to convergence and to accommodation, are associated impulses; and it is owing to there being great individual differences in the extent to which they can be dissociated—differences which may be partly acquired and partly inherited—that a degree of accommodation can be maintained by one hypermetrope without asthenopia and without the sacrifice of binocular vision, which in another hypermetrope to the same extent would be impossible for any length of time. Certain cases of periodic squint show, indeed, how close some have to approach to the limit of their power of dissociating the two impulses. If they happen to have equal and good visual acuity in both eyes, the desire for binocular vision, or what is called the impulse to fusion of the retinal images of the two eyes, is sufficiently strong to effect the dissociation under favourable circumstances, that is, when the nervous centre is in a vigorous state. But the maintaining of the fusion gives rise to asthenopia, and brings about a tiring of the nerve centres, which

renders the continuance of the effort impossible; and it is every now and then (sometimes at such regular intervals as to have been looked upon as of the nature of ague by some writers) relinquished for the alternative of binocular vision with convergent strabismus (mostly with diplopia), to be resumed again after the period of rest which this state affords. Besides cases of intermittent squinting occurring on alternate days, I have met with an interesting illustration of this fact in the case of a young girl of twelve, who began to squint and see double nearly every afternoon, and was in the habit of getting rid of this distressing symptom by going to sleep for an hour or two. On correction of her hypermetropia these symptoms disappeared. Every now and then one sees cases, too, of strabismus occurring suddenly after debilitating illnesses, poisoning of the nerve centres, or lassitude from any cause, which can only be explained by assuming that the tendency to fusion, or what has been called the breadth of fusion, has been diminished; and in such cases the dissociation of the impulses to accommodation and convergence has been constantly maintained hitherto generally with more or less asthenopic pain. In them, although binocular vision for near objects is effected with difficulty, the difficulty is, either from great existing breadth of fusion, or from the less extensive or constant call made on the store of power at the disposal of the individual, not sufficiently great to necessitate their giving up the advantages of binocular vision until something unusual occurs, such as already signified, to weaken the nervous centres. A much more common cause of sudden squinting is ulceration of the cornea, the obvious explanation of which is the previous existence of predisposing causes, which immediately assert themselves when the value of binocular vision, from defective vision of the one eye, becomes lessened. When this takes place in adults, there has usually been asthenopia previously, though not always, as the squinting position may be due merely to the assumption of the position of muscular equilibrium, otherwise easily departed from in the interest of binocular vision.

The pain complained of in accommodative asthenopia is not generally very severe, and is more of a tiring nature. It is

referred sometimes to the eyes, but more commonly to the supraciliary regions of the forehead, sometimes to the whole head.

The obvious indication for treatment of accommodative asthenopia is to assist the accommodation by the use of convex glasses; and for this purpose, in cases of hypermetropia, it will generally be found practically sufficient to correct the manifest portion, enjoining, however, a constant use of the suitable convex lenses, both for distant and near vision. An attempt to correct the total, or nearly the total, amount of the hypermetropia is often unsuccessful, as, the spasmodic tendency to accommodation which exists in hypermetropic individuals, the more forcibly the younger the age of the individual, produces a combination with fully correcting glasses equivalent to myopic refraction, and the indistinctness of vision thus caused leads frequently to the laying aside of the glasses altogether—distinct vision, even with asthenopia, being preferred to short-sight and no pain. But in addition to the short-sight thus induced, it not unfrequently happens that the full correction, though theoretically calculated to relieve the symptoms of asthenopia, fails to do so, in which case the individual is altogether worse off with, than without, his glasses. The fact is, that accommodative asthenopia, though sometimes disappearing at once under the use of proper convex lenses, is often at first little influenced by the altered and improved conditions which they bring about; there is, as it were, a sort of neurosis set up, which only slowly disappears under the more favourable conditions. Sometimes, indeed, the cause of the pain from the first has not been any accommodative difficulty, though the existence of hypermetropia has justified such a suspicion, but has been more of the nature of nervous asthenopia.

Muscular Asthenopia.—Pain primarily due to over-exertion of the ocular muscles consequent on abnormal attachments, deficiency of power, or abnormal positions of muscular equilibrium of the two eyes, is certainly much less frequent than was at one time supposed. We may, however, include under the head of muscular asthenopia the pain or discomfort which arises from two condi-

tions: 1. A defective impulse to convergence, or, as it is sometimes called, insufficiency of convergence; 2. A *high* degree of latent divergence or convergence of the visual axes, which necessitates the keeping up of an abnormal strain on the antagonistic muscles in the interest of binocular vision. Insufficiency of associated movements other than convergent are, as far as I have observed, not associated with asthenopia. I have met with three curious forms of paralysis of muscles used in associated movements, unaccompanied by asthenopia. In one case, complete paralysis of both interni; complete paralysis of both externi, in one of which the condition remained unaltered for four years, and the patient eventually died insane; and, lastly, two cases of what has been called by Hutchinson ophthalmoplegia externa. The cases cited by Hutchinson are of two distinct origins: one depending upon gross cerebral changes, possibly of a syphilitic nature, and not so very rare; and the other to which I refer depending on localized degenerative cerebral lesions. An insufficient impulse to convergence may be of the nature of a true associated paralysis—paresis of convergence. Such cases are certainly uncommon, but I have seen several, all giving rise, owing to the effort required to avoid diplopia, to asthenopia. But much the most common cause of convergent insufficiency, often wrongly called insufficiency of the interni, is the defective impulse which arises from want of support from the associated impulse to accommodation met with frequently in the higher degrees of myopia. In these cases sufficient convergence for the requirements of binocular vision is with difficulty maintained, owing to the distance from the eyes at which vision becomes distinct approaching much nearer the extreme limit in which the two axes can be converged than under normal conditions of refraction. The condition is exactly the converse of that described as causing a strain in hypermetropic individuals; there is in both cases an abnormal dissociation of the two associated impulses in the interest of binocular vision; but whereas in hypermetropes the accommodation is strained without corresponding convergence, in myopes the convergence is exerted independently of accommodation. And here again we meet with great individual

differences in the amount of asthenopic trouble to which the necessity for dissociating the impulses to convergence and accommodation gives rise—differences which are partly acquired and partly hereditary. A person who becomes rapidly highly myopic, or a myopic individual whose more immediate progenitors have not suffered from the same abnormal state of refraction, is more likely to experience asthenopic difficulties than one in whom the myopia has only been slowly progressive, and in whom it is inherited from perhaps a good many generations of ancestors. There is, however, a tendency to the abnormal relation between convergence and accommodation necessarily existing in myopes with binocular vision and uncorrected refraction becoming confirmed, just as in hypermetropes there is a portion of the hypermetropia rendered latent by involuntary ciliary contraction; and this circumstance explains why it is often difficult, or even impossible, for myopic individuals to wear at first the glasses which fully correct their refraction, more especially if the first correction is made pretty late in adult life. It is easy to satisfy one's self that the smaller power of convergence in many cases of myopia is not due to any weakness of the interni muscles, and therefore, as already remarked, wrongly expressed by the term insufficiency of the interni; for we find that the associated movements to the right and left are effected with the greatest ease, to an extent which calls for a far greater deviation inwards of either eye than is possible when attempts at convergence are made.

The nature of the pain in this kind of muscular asthenopia is much the same as in accommodative asthenopia. It is referred sometimes to the eyes, but more frequently produces a diffused tired feeling in the forehead and other parts of the head.

Asthenopia, again, which is not accommodative, or muscular in the foregoing sense, may nevertheless be assumed to be connected with the muscles when we find any *very considerable* degree of latent deviation of the axes, or when as a congenital defect or as the result of operation, an abnormal position of attachment necessitates, under all circumstances, a very much greater muscular action in order to bring about a certain position of the eye. I say very considerable, because one often sees latent divergence where

there is no asthenopia; and it is only when the point of departure, so to speak, of the action of the interni is very much displaced that this condition is likely to give rise to difficulties. In latent convergence the difficulties are greater, owing to the smaller tendency altogether to deviation of the axes of vision. A latent convergence must increase the difficulties of a hypermetrope, and is one of the factors which (along with monocular amblyopia, insufficient breadth of fusion, and in many cases hypermetropia) lead to convergent strabismus. Conversely, a hypermetrope, whose eyes in their position of equilibrium[1] would diverge, is less likely to be troubled with asthenopia. In some cases, then, a condition which might be supposed to give rise to muscular difficulties, and consequently asthenopia, acts rather in a manner tending to counteract asthenopia.

The pain in what might therefore be called true muscular asthenopia, as being due to muscular fatigue, and not strain on the nervous mechanism called into play in associated muscular action, is most frequently referred to the eye itself, giving rise to a tired, aching feeling of the eyeball.

The indications for treatment in muscular asthenopia are for the form depending on insufficiency, strengthening of the nervous impulse to convergence, or occasionally to divergence, either by the selection of suitable concave or convex glasses, or, in the rarer cases, where the cause is more of a true paretic nature, by the use of means

[1] By the position of equilibrium is meant the position of the two visual axes when one eye is fixing an object at a distance (without exertion of accommodation), or staring vacantly into space, whilst the other is occluded. The position of equilibrium is not easily determined with certainty in hypermetropia, because of the accommodation brought into play even when fixing a distant object. Under atropine, too, there must be, when an attempt is made to see anything distinctly at a distance, an *impulse* to accommodation, with which a degree of convergent movement is naturally associated, so that the position of the occluded eye is not that of true equilibrium. Thus parallelism of the axes, when one eye is occluded and the other fixing a distant object, may, and often does, mean divergent equilibrium; and convergence under similar conditions does not necessarily imply a convergent position of equilibrium, which may be parallel or even divergent.

calculated to restore, if possible, the vigour of the nervous centres; it is seldom that any operation is called for or likely to do good. On the other hand, the more truly muscular cause of asthenopia may be obviated by suitable operative measures, the most successful of which is the advancement of the weakened or disadvantageously placed muscle. It is not always, however, that this treatment which seems to be indicated is effectual in removing the pain, notwithstanding the apparent[1] or real improvement to be found in the muscular relations. This is no doubt owing to the neurosis set up by the long continued previous conditions, and which cannot disappear at once; or it may be owing to the asthenopia being purely nervous, and unconnected with those muscular abnormalities which might be supposed to give rise to it.

Retinal Asthenopia.—Pain in the eyes, often described as right at the back of the eyes, unconnected with any accommodative or muscular abnormality, and coming on, often very severely, at longer or shorter intervals after use of the eyes for reading, etc., is extremely common. Often the condition is associated with more or less sensitiveness to light. In many, and indeed most, of these cases there is absolutely nothing to be found in connexion with the eyes, at any rate, to account for the persistence or severity of the symptoms. Either the strain on the attention becomes soon too overpowering, or the retina itself is hypersensitive and easily tired; at all events there is some weakness in the tone or capabilities of the nervous mechanism of vision. Such a state of nervous asthenopia is probably frequently of reflex origin, though the region from which the afferent stimulus proceeds is not often known. It is more common in women than in men, which has

[1] I have said apparent as well as real, because it often happens that the rest afforded by the operation is sufficient to permit of a greater momentary exhibition of muscular power than could be elicited before; and, besides, there is a considerable personal error in the estimation of the power of a muscle, more especially if use be made of any instrument, such as a dynamometer, from which one expects accurate results, just as there is a possible personal error in the estimation of refraction by means of the ophthalmoscope, or in the determination of the exact limits of peripheral vision.

led some authors to ascribe it to uterine irritation, and even to describe a particular form of chronic inflammation with which it is associated. The causal connexion between the two, as a matter of constant or even frequent occurrence is, however, in the highest degree improbable. Intestinal irritation also appears sometimes to give rise to this form of asthenopia. In a great number of cases—and I am disposed to think in by far the greatest number —the pain is due primarily to over-exertion of the eyes at a time when, after a debilitating illness, or a state of malnutrition, anæmia, etc., the nervous tone is below par. The pain thus originating becomes chronic, a sort of neurosis or habit, and may become so distressing as to render reading impossible for more than a few minutes at a time. Sometimes the condition of the eyes themselves is such as to induce asthenopia by over-use, such as congenital amblyopia and astigmatism, where the difficulties in deciphering small type are considerable, and therefore accompanied by abnormal strain. Indeed, one can bring on the same kind of feeling in one's own eyes by attempting to read small type either beyond the limit of distinct vision, or with an illumination barely sufficiently powerful for the purpose; and that independently of over-exertion of convergence or accommodation. Individuals suffering from this form of asthenopia generally take, sooner or later, to the use of blue or dark spectacles; and by so doing merely increase the sensitiveness of their retinæ. Such spectacles should only be employed in cases of deep-seated inflammation, in which the tempering of the light passing into the eyes is one important means of complying with the indication for rest, or under conditions of excessive glare from powerful sources of illumination or reflexion.

Many cases of retinal or nervous asthenopia are amongst the most severe and rebellious that one is called upon to treat. Cold-water douches, and iron internally, are indicated in some cases; others are much benefited by hot sponging or fomentations; and when there is absolutely nothing abnormal to be discovered in the eyes, an energetic attempt should be made to break the habit of giving up reading as soon as the pain comes on. This can only be done gradually, but is successful in many cases.

Pain independent of the use of the eyes is often complained of, and is generally of a neuralgic character, and more or less intense. The diagnosis is easily made when there are points of special tenderness on pressure round the eye. Often the eyeball itself is tender to pressure. Occasionally, no doubt, the pain is reflex, but the origin of most cases is a hypersensitiveness of the supra- and infra-orbital branches of the fifth nerve, caused generally originally by exposure to cold. Keeping the head warm, especially at night, and the use of iron, arsenic, and quinine, is the line of treatment indicated. In rare instances an intraocular tumour may give rise to pain before having produced inflammatory or visual changes, to be detected on superficial examination, though hardly before causing some limitation of the field of vision.

Inflammatory Pain.—Of pain produced by inflammation, or by some definite lesion other than inflammation, there are two pretty distinct types: one in which the pain becomes much more severe under the influence of light, in which there is then more or less marked photophobia, and the other in which photophobia is absent. The co-existence of photophobia with the irritation of sensory nerve filaments in the eye is an instance of reflex, or it may be direct transference of stimulation from a nerve of common to a nerve of special sensibility, which, so far as I am aware, has no absolute parallel anywhere else in the body. There is no reflex in the ordinary acceptation of the term; that is to say, the irritation of fibres of the fifth nerve does not lead to stimulation of those of the optic nerve so as to give rise to subjective light sensations, but the transference is of such a nature as to cause the functional activity of the optic nerve, which under ordinary circumstances is painless, to be associated with pain, sometimes very intense. We do not find that irritation of the sensory fibres supplying the more external parts of the ear or nose give rise to painful hearing or smelling. Certainly a very loud or shrill sound, or even a moderately loud sound, after a prolonged period of silence, produces a painful or disagreeable sensation which causes one more or less involuntarily to stop up one's ears, just as excessively strong light, or a light which, when the retina has become accustomed to it, is easily

tolerated, when falling on the eyes after a period of darkness, is painful to the eyes, which therefore close against it. So far there is a parallel, but there is not the same hypersensitiveness induced, and accompanying functional activity of the auditory nerve, so soon as there is an unwonted stimulation of a few superficial twigs of the nerve supplying the ear with common sensibility, as is the case with the optic nerve immediately after some disturbance of or loss of superficial corneal epithelium. It is difficult to account for this sensitiveness only caused by light, as the optic nerve itself is apparently not very sensitive; there is, at all events, no pain caused by inflammation of the optic nerve, and stimulation of the cut end after enucleation of the eyeball is generally hardly felt. On the other hand, we have seen in discussing nervous asthenopia that, under various circumstances not absolutely pathological, the mere use of the eyes is accompanied by pain, so that it is not easy to avoid the conclusion that somewhere or other, possibly in the retina itself, there is an intimate connexion between the optic and fifth nerves, whether acting through the vaso-motor system or in what manner can at present only be conjectured. Occasionally the increased irritation of sensory nerves caused by the action of light is spread over a wide area, one curious example of the wide diffusion being the sneezing which is frequently met with in children on forcibly opening their eyes, firmly and persistently closed from photophobia. The irritation also causes an immediate hypersecretion of tears, photophobia being always accompanied by lacrimation.

The conditions giving rise to photophobia are generally some lesion or inflammation of the cornea, iris, or ciliary body, though the degree of photophobia varies much even in apparently similar conditions. Traumatic, phlyctenular, and interstitial keratitis and iridocyclitis are the most frequent conditions in which we meet with more or less photophobia. Deep and extensive ulcers of the cornea are not so much complicated by this symptom, though often giving rise to great pain, probably because they destroy without keeping up a prolonged irritation of the superficial nerve endings in the cornea. Some of the most intense cases of

pain as well as photophobia accompany slight abrasions of the cornea, caused, for instance, by a scrape from a child's nail, or a twig of a bush or tree, or in some other way; and there is a tendency for the irritation thus set up to give rise to recurrent attacks at intervals, in which the pain is often quite as severe as at the time of the injury. Photophobia once set up is often maintained, more owing to the sensitiveness of the retina which follows prolonged exclusion from light (as in retinal asthenopia by the constant use of dark spectacles or residence in darkened rooms) than to the exciting cause of the local stimulus. This is especially the case with strumous children, who have been suffering from superficial (phlyctenular) keratitis, where the photophobia is often seen lasting long after there is any sufficient cause to account for it on any other supposition. Here it is that efforts to break the habit, such as the shock to the nervous system by immersing the head in cold water, the forcible opening of the eyes, or the use of atropine or eocaine, are often almost immediately efficacious. So that although photophobia may be primarily a provision of nature for the protection of the eyes, it often becomes a distinct pathological condition, owing often to misguided treatment, actuated as a rule by over-anxiety on the part of parents or others. The action of atropine, in often diminishing pain and photophobia, is mostly due to its direct anodyne effect,—to some extent, however, and more especially in interstitial keratitis and other deep inflammation to paralyzing the movements of the iris.

The pain of iritis is often very intense, and affords a good guage of the severity of an attack. It is generally most intense at night, or in the early morning, and radiates over the region supplied by the supra- and infra-orbital branches of the fifth nerve, being often complained of as at the side of the nose. Although the accompanying photophobia is rarely as intense as in some more superficial inflammations, the withholding of light renders the pain less severe by keeping the iris at rest, and this, along with the use of a mydriatic for the same purpose, is the most important indication for local treatment, both as regards the alleviation of pain and

the avoidance of complications which may afterwards be sources of greater or less danger for the eye. In purely rheumatic forms the pain is often checked at once by the use of salicylate of soda internally, given frequently (7–8 grains every hour).

In simple iritis there is no particular sensitiveness of the eye to touch. When this exists in an attack of iritis, it indicates a participation of the ciliary body in the inflammation (cyclitis); and the degree of sensitiveness, sometimes intense, affords the only sure indication of the severity of the accompanying cyclitis.

In simple choroiditis, except in cases where the intraocular tension becomes distinctly increased, there is but little pain or photophobia; sometimes a dull aching pain at the back of the eye is complained of. Suppurative choroiditis, leading to panophthalmitis, gives rise, however, to dreadful suffering, the intensity of the pain being, as far as inflammatory conditions of the eye are concerned, only equalled, if at all, by that of acute glaucoma or of acute inflammation of the orbital tissues; but this is mostly due to the participation of other tissues in the inflammation, and particularly to the tension in the eye caused by the exudation.

In cases where there is considerable inflammatory œdema of the parts about the eye, the position of the greatest pain on pressure is a point of importance in the diagnosis, as in this way we may distinguish between periostitis of the margin of the orbit and inflammation of the lacrimal sac.

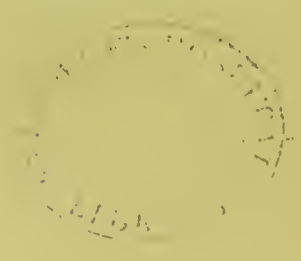
The pain of extensive or deep ulcers or abscesses of the cornea, and especially that form of septic inflammation which is accompanied by a deposition of pus in the anterior chamber (hypopyon), is often very severe, as is the case always where suppuration takes place in tense tissues. The relief of tension caused by opening such a septic abscess, though eventually causing great relief of pain, produces at the time often the most agonizing suffering.

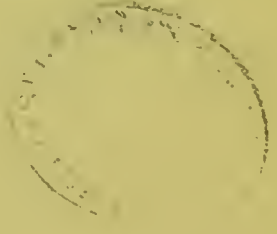
The pain of glaucoma is very variable, just as are the symptoms of that disease altogether. It is probably greatly due to irritation of the sensory nerves from the accompanying increase of intraocular tension, though increased tension alone is not always associated with pain. In acute inflammatory glaucoma the irritation

of the fifth nerve is so great as to extend to all its branches, and gives rise to such severe general symptoms as to be apt to divert attention from their original source and lead to errors of diagnosis. Such a mistake is all the more likely to occur when there has previously been blindness of the eye from which the pain proceeds.

One of the commonest, and at the same time most obscure, sources of pain in the eye—seldom, however, except when acute, very severe—is conjunctivitis. In the acute forms it is a sharp burning pain, very different from the diffuse neuralgic pain of iritis, and giving rise at the same time to the sensation of there being dust or sand in the eye. When chronic, it is more a dull aching pain which is complained of. This is often due to the slight glueing together of the palpebral and ocular conjunctiva by the glutinous secretion which is so common in many cases, and in these cases the lids feel heavy and tired. Frequently, though, there is no heaviness of the lids arising in this way, and it is often difficult to imagine how the slight amount of visible inflammation should be accompanied by so much discomfort. In this respect, moreover, there is a vast difference in different individuals, a difference greatly due to the sensitiveness and occupation of the individual. A degree of conjunctivitis, which in a labourer would be quite disregarded, will often cause great distress to a lady who, besides being naturally more sensitive, has more leisure to brood over her misfortunes.

From the preceding short sketch of one of the most common and important subjective symptoms met with in affections of the eye, it will be evident that the existence of pain will often lead one to the detection of pathological conditions which might otherwise escape notice, owing to the absence of other striking symptoms; and that, besides, the nature and severity of this symptom may be of use in leading one to the diagnosis of the site of, as well as the course taken by, inflammatory diseases of the eye, even though it may in many cases be of less importance than the accompanying objective signs.





CHAPTER II.

DIPLOPIA—POLYOPIA.

When double vision is complained of, we have, in the first place, to determine, by covering first one eye and then the other, whether in either case it is still present, or whether it always disappears when one eye alone is used. We have therefore to distinguish between binocular and monocular diplopia.

Binocular Diplopia.—In order that there should, under any circumstances, be binocular diplopia, it is necessary that the following three conditions should be complied with:—1. That there should be a fair amount of vision in both eyes; 2. That no circumstance should have led to a more than physiological suppression by the mind of the image falling on either eye; and 3. That both the visual axes should not be directed simultaneously on the object which engages the attention. (There is a curious possible though rare exception to this third condition, which will afterwards be referred to.) Thus there may be no diplopia complained of, even though there be wide divergence of the visual axes, owing to the suppression of the image of the one eye from more or less monocular blindness, or more frequently from habit. This is the most common condition in ordinary cases of concomitant squint. In such cases the suppression of the image of the one eye, when the other is used for fixation, is sometimes so complete that under no circumstances can it be made apparent; in other cases, by holding a red glass or a prism with the angle directed upwards or downwards in front of the fixing eye, the faulty image of the other is at once seen, showing that the suppression is only effected for normal conditions of similarity in the optical images, as well as for a retinal area only, on which, under ordinary circumstances, the images corresponding to those occupying the centre of the retina of the fixing

eye are received. Again, an individual may complain of diplopia even although the visual axes are capable of crossing, and actually do cross on the same object. This is the case when other objects than the one fixed engage the attention, and is, in fact, physiological in so far as all objects not directly looked at are seen double. When, however, this kind of double vision is complained of, it is owing to an abnormal degree of attention being directed to objects other than those fixed. Usually there is a suppression of one of the images of other objects than the one on which the visual axes are directed,—a suppression which is often so complete as to render it difficult for many people to become conscious of this physiological diplopia. Which eye is the one whose images are suppressed in any particular case may be determined by a very simple experiment. By asking any one, while keeping both eyes open, to hold up their finger in a line with some distant object, and then close first the one and then the other eye, they will generally find that the finger exactly covers the object as seen by one eye, while it deviates to one side when looked at by the other. This shows that only the image of the eye in a line with which and the distant object the finger has been placed is observed, that of the other being more or less completely suppressed. The cases of diplopia just referred to are due to the opposite condition, viz., to a too ready appreciation of the physiological double images. I have mostly met with this in women, but occasionally also in men. When once discovered by them, and not recognised as physiological, they contract a habit of directing attention to the double images, which sometimes becomes almost painful. Occasionally it is the nose which is in this way brought prominently before their notice, and which always appears to get in the way of other objects, by attracting attention at the same time; at other times, any objects both beyond and within the point of fixation are continually forcing their two images on the attention. This condition is by no means easily got rid of, even when the cause has been explained to the patient.

Patients who suffer from true diplopia, due to a misdirection of one of the visual axes on the object fixed, generally complain most of the doubling of that object, one image of which

they usually recognise as the "true" image, that is, belonging to the properly directed eye, and the other as the "false" image, the image of the misdirected eye. The false image not only occupies a distinctly abnormal position, but is, besides, more or less indistinct, owing to its corresponding to a peripheral, and not a central, retinal impression. Although, however, we can infer from observing to which eye the true image belongs, which is the fixing eye, it does not by any means follow that the defect in muscular activity is to be found in the other, as the cause of diplopia may be a condition common to both eyes, or confined to the fixing eye alone, the eye made use of for fixation being often determined by causes altogether unconnected with the condition of which the diplopia is a manifestation. Occasionally, whilst recognising the doubling of the object fixed, the more distressing symptom complained of is a veiling of that object by the projection over it of the image falling on the macula of the misdirected eye, so that two different objects are constantly seen, the one through the other, as it were, just as the two images of a stereoscopic picture are projected to the same position in space. In cases where diplopia is complained of, it is often not constant, or it may be not present under all circumstances. This may be due to want of constancy in the conditions giving rise to it, as is the case in periodic squint, when the two visual axes are at certain times both directed on the same objects, whilst at other times only the one is properly directed; or there may be frequently, suppression of the image of the misdirected eye. Again, in many cases of paralytic squint, especially where the paralysis is of recent occurrence, and not associated with contractions of antagonistic muscles, there is double vision only in certain directions of fixation. In periodic squint it is, indeed, greatly owing to the presence of diplopia during the period of squinting that the squint is not constant, efforts being made to overcome the muscular difficulties in order to avoid the disagreeableness of double vision, even although the alternative is often asthenopia. In ordinary cases of concomitant squint with diplopia, fortunately a not very common complication, there is double vision over the whole extent of the field of fixation, the degree of

separation of the double images being much the same, though not exactly the same, for all positions of objects equidistant from the eye.

In cases where there is a defect of convergent power, or an increased tendency to or spasm of convergence, there is often diplopia within or beyond a certain distance from the eye, a distance which not only varies according to the degree of the abnormality, but also according to the position of the eye above or below the horizontal plane of fixation. Thus any weakness of convergence reveals itself more readily when the eyes are directed upwards, and therefore gives rise to diplopia for objects further away from the eyes than in the case of fixation in the horizontal plane; whereas, when the eyes are directed downwards, the defect is less appreciable, and diplopia is only evident on approaching the object nearer to the eyes than is required under similar circumstances to produce double vision in the case where fixation takes place in the horizontal plane. On the other hand, when there is either a nervous spasm of convergence, a very rare condition, or an acquired preponderance of convergent over divergent movements, as in some cases of myopia, the exact opposite is the case, that is, the abnormal conditions are most marked when the eyes are directed downwards, and least so when directed upwards, so that there is under such circumstances a greater range through which the diplopia is manifest for the depressed than for the elevated plane of fixation. This is owing to the circumstance that there is a greater tendency towards convergence on looking downwards than on looking upwards. Thus, while it is possible for many to diverge their visual axes on looking upwards so as to see distant objects, such as stars, in crossed double images, this is not only impossible when the eyes are directed downwards, but in that position there is even a much greater reluctance in relaxing the degree of convergence assumed for seeing objects near the eyes, so as to overcome, by a divergent movement, the diplopia which a prism with the angle outwards held in front of one eye causes. It is probable that these differences are the result of the requirements of vision; objects when fixed considerably below the horizontal plane through the eyes being usually near, and therefore

necessitating convergence, those above that plane, on the other hand, being mostly distant.

It is customary, in making examinations with a view of determining the relative positions of the double images in cases of diplopia, to select nine positions for that purpose, five of which are called primary, and four secondary positions. The primary are those into which the eyes are brought by purely lateral or vertical movements, whilst the secondary positions are such as result from movements which are combinations of these two. These combined movements cause the vertical and horizontal meridians of the eye to be rotated outwards or inwards; in other words, the movements bringing about the secondary positions (upwards and inwards, upwards and outwards, downwards and inwards, downwards and outwards) are associated with a torsion or twisting of the eyes round their antero-posterior axes. The tests are generally made with a lighted candle, held first in the horizontal plane, at a distance of six to eight feet straight out from the face, then on the same level some distance to either side, then in the middle line above and below, and finally above and below to either side. The examination is facilitated by holding a red glass in front of one eye, as this not only makes the double image more apparent to the patient, but lets the observer know at once to which eye each image belongs. As a general rule, it is best to hold the coloured glass in front of the eye used for fixation, or the one in which there is the greatest visual acuity. In this way important information is gained as to the nature of a paretic affection, which is often revealed by the diplopia before it could well be detected by observations based on objective methods of examination.

In paralytic squints, the positions for which the diplopia exists are those into which an attempt is made by the weakened muscle to bring the affected eye in association with the other eye, so that the separation of the double images is greater according as such positions of the object seen double, are more and more in the direction of the action of that muscle, and, of course, also in proportion as the paresis is more and more complete. An exception to this rule is found in cases where there is a paresis in both eyes

of two or more muscles used in associated movements. Besides the differences in the relative separation of the double image which are found to exist for these different positions in the various forms of paresis, there is a difference in some cases due to the greater or less tendency to convergence, already referred to, according to the degree of elevation of the eyes. Thus, in cases of paresis of the sixth nerve, we find, owing to this circumstance, that the line of demarcation between single and double vision, instead of being vertical, passes obliquely from the side of the affected eye above to that of the sound one below.

When the image seen to the right belongs to the right eye, or that seen to the left to the left eye, the diplopia is said to be *homonymous;* on the other hand, when the right image is that of the left eye, and the left that of the right, the diplopia is *crossed.* Homonymous diplopia is present when there is relatively too great convergence of the visual axes compared to the distance of the object fixed; crossed when there is relatively too great divergence.

Homonymous diplopia is met with in paresis or paralysis of the sixth and fourth nerves, in the rare cases of isolated paralysis of the inferior oblique, and in strabismus convergens, also in cases of spasm of convergence. In a large proportion of cases of convergent strabismus, the suppression of the image of the squinting eye is so complete as to exclude diplopia, but there is always diplopia in cases where the strabismus has come on suddenly, or in adult life, as when associated with myopia, and sometimes hypermetropia, or when due to any cause at the same time unconnected with a loss of distinct vision in the one eye. The diplopia arising from convergent strabismus met with in myopia is, as has been already said, often only present for objects beyond a certain distance from the eyes, a distance which tends to become closer and closer in the course of time, and in this respect it differs from ordinary cases of hypermetropic strabismus, where, when there is diplopia, it exists for all distances. Occasionally one sees cases of hypermetropic squint, however, with diplopia, presenting exactly the same characteristics as that associated usually with myopia, where distinct distant vision is impossible for any time, at any rate

without diplopia, whereas there is always single vision within a certain distance. These cases are at once cured by the use of correcting glasses, whereas it is only at the outset that the corresponding myopic strabismus can be overcome without tenotomy of the internal rectus muscle. Sudden convergent strabismus is occasionally met with as a result of central causes which lead to diminution in the breadth of fusion, and always gives rise to homonymous diplopia.

Crossed diplopia occurs in paresis or paralysis of the third nerve, and in some cases of divergent strabismus; or, within a certain distance, in paresis or insufficiency of convergence.

If we fix a distant point, we observe that the crossed double images of a nearer object are further and further apart the nearer the latter is to the eyes, and similarly the homonymous double images of a distant object separate more and more the nearer the object is approached on which the eyes are fixed. There is, in fact, a definite angular separation of the two images corresponding to the distance from the macula of each eye on which the retinal impression takes place, although the distance of apparent or linear separation depends on the position of the plane on which the two images are mentally projected. We might, therefore, expect that it would be easy to calculate the angular deviation of the visual axes under any circumstances from a determination of the distances separating the double images. This is, however, not always the case, though it generally is, and for a reason which is not quite so simple as might be at first supposed. The outward projection of images formed on the macula of the eye depends apparently on the knowledge we have of the position of our eyes gained by the constant experience of the effort required to bring about that position, so that when a muscle becomes suddenly weakened, and a greater effort has to be made to attain, or endeavour to attain, some particular position, this increased effort is associated with a displacement of the line of projection in the direction of the action of the weakened muscle. Hence it is that, as the projection of peripheral images is always the same relatively to that of the central ones, there is a displacement of the projection

of the peripheral impression of the deviating eye in the same direction and to the same extent as if that eye were properly directed on the object fixed by the other. It is this abnormal projection of the images of the paralyzed eye which gives rise to the giddiness, etc., experienced by patients with diplopia due to paralysis. The faulty projection of retinal images caused by an abnormal stimulus being required to bring the eye to bear on an object is well illustrated by the following experiment due to Helmholtz: Prisms of 16°–18° are placed in front of both eyes with the bases both turned the same way, so as to produce a lateral displacement of objects. On grasping suddenly at any object we find that we grasp too far to the side to which it is apparently displaced. After a few trials this is corrected for, and on removing the prisms and attempting quickly to seize the object, we then grasp too far in the opposite direction.

Another circumstance of a somewhat similar nature may, however, produce the effect of causing the separation of the double images to differ in extent from that which the position of the visual axes would seem to demand. Thus it sometimes happens that after operations for convergent strabismus, even although slight convergence is still left, there is a spontaneous complaint of diplopia, or diplopia may easily be elicited by holding a red glass in front of one eye, when the double images are found to be crossed instead of homonymous. In such cases there must have been in the previous position of the eye a projection of the faulty image on or towards the point fixed, and therefore of the image falling on the macula lutea inwards, *i.e.*, to the opposite side of that point, whereas more commonly, notwithstanding the misdirection of an eye, the image of any point falling on the macula lutea is projected to the same place in space as that occupied by the object fixed, though, as we have already seen, there is only occasionally a consciousness of this projection. A new visual axis is, in fact, acquired which passes through some peripheral point of the retina occupied by the image in the misdirected eye of the object fixed by, and therefore forming an image on the macula lutea of the other, so that the correction effected by the operation,

producing as it does an outward displacement of this new visual axis, introduces a condition corresponding to divergent squint. I have lately met with a curious case of anomalous projection and diplopia which illustrates this point.

W. T., aged 30, had, when admitted into hospital, divergent strabismus, which had existed for many years. The right eye diverged $V = \frac{20}{50}$ in both eyes. In the left it was improved to $\frac{20}{30}$ with + 1·50 cyl. The right eye was not improved by any glass. There were no abnormal ophthalmoscopic appearances. He complained of double vision, which had come on nine months previouly at a time when he was greatly prostrated by starvation, and which had persisted since. On examination, notwithstanding a divergence of about 3‴, the double images at a distance of 4′ from the eyes were close together, not much more than 1″ apart, and *homonymous*. I performed tenotomy of the right external rectus and advanced the internal rectus (Schweigger's operation). The result, a fortnight afterwards, was almost complete parallelism for distant fixation with good abduction power, certainly no marked insufficiency of the externus, and considerably increased power of adduction. The diplopia was still marked, and the double images much further apart than before operation (about 10″ at 4′), and homonymous.

The only possible cause of the sudden occurrence of the homonymous diplopia in this case, notwithstanding the long existence of the divergence, could be the supervention of a paresis of the sixth nerve, not improbably caused by exposure to cold and starvation. The fact, however, of the diplopia being homonymous shows that the projection up to the time of the paresis was anomalous in the manner referred to, *i.e.*, was in accordance with the position of the eye and not with the normal conditions, so that the relative convergence which the paresis produced in the direction of action of the external rectus gave rise to the same effect as an absolute convergence would have done had the original position of the visual axes been parallelism. The long continuance of the diplopia, in spite of the short distance separating the double images, and the apparent disappearance of the paresis, points on

the one hand to an alteration in the tone of the external rectus, and on the other to an absence of any power of fusion of double images. The more pronounced homonymous displacement of the double images after operation was evidently due to the persistence of the previous conditions of projection. The eye, although having a different position of equilibrium, continued to project its retinal images as if it stood in the previous position, the knowledge of which it had, so to speak, acquired just as more frequently a deviating eye continues to project in a manner as if it were properly directed.

This abnormal cause of diplopia does not, however, usually last long, as a new point on the retina soon becomes identified with the macula of the fixing eye, or the suppression of the image falling on the misdirected eye soon becomes complete. In this way it may happen that although the centres of each eye are directed on the same object there may yet be diplopia. This is the exception referred to as rendering the third condition for diplopia not universally true.

The directions in which the faulty image may be displaced are of three kinds:—1. Purely vertical or lateral. 2. Sloping. 3. In its apparent distance from the eye. The lateral and vertical displacements are the direct consequence of the imperfection in association of the lateral and vertical movements of the two eyes. They are therefore met with in all the positions in which these associated actions should come into play. The sloping displacement, in which the faulty or the true image appears to slope towards or from the other, is due, in the case of paresis of the rectus externus or rectus internus, to the imperfection in the association of movements necessary to attain the secondary positions, in the case of the isolated paresis of other muscles, to the absence as well of their co-operation in the vertical movements, which can only be properly effected by the combined action of a straight and oblique muscle. The difference in the positions of the vertical and horizontal meridians of the two retinæ, brought about by efforts to attain these positions under the abnormal circumstances, causes the apparent want of parallelism between the images. Owing, however, to the secondary positions

being less natural and more constrained positions, there is not the same degree of precision in the association of the two eyes ever acquired in attaining them as is found for the primary positions. There is, consequently, when one muscle which aids under normal conditions in bringing about a secondary position is weakened, a position of the false image which corresponds not only to the defective action of that muscle, but also to the incomplete attempts at associated action of the remaining muscle or muscles, which results in bringing about a degree of apparent sloping of the image which does not always correspond in amount, and is generally greater than that which, from the conditions of physiological action, might be expected. The third displacement, that which causes the false image to appear nearer or further away from the eye than the true one, is one depending upon the position in space to which it is projected, and on this account is by no means invariably the same, even in the same case. The monocular estimation of distance is very imperfect, and easily affected by many circumstances, but there seems to be a tendency for images falling on the retina above the macula lutea to be referred to nearer distances, while those occupying positions below the macula are more readily projected to greater distances. This is probably due to the experience gained under the ordinary conditions of binocular vision. If we fix any object on the ground, the images of nearer objects on the same plane are formed on higher points of the retina. But it is impossible to draw conclusions as to monocular projection from what obtains binocularly, where the conditions for the estimation of distance are so much more favourable. Still we may, by simply holding a prism with the angle downwards in front of one eye, convince ourselves that the image of that eye is nearer to us than that of the higher one belonging to the other eye, provided always that the object fixed does not lie on a vertical plane, or on one sloping away from the eyes, otherwise its image being projected on a plane in such a position appears to be more distant.

A description of the relative positions of the double images in paralysis and paresis of the different muscles alone, or in various combinations, does not form part of the object of this sketch of

diplopia as a symptom of ocular disorder; such a description is to be found in most text-books on ophthalmology. Many of these pareses can only be made out by careful examination of the relative positions of the double images. I have met with isolated pareses of all the muscles except the inferior oblique, and with many different forms of combined paresis, some of which require a good deal of consideration before the correct diagnosis can be arrived at. The more common cases of paresis of the sixth, third, and fourth nerves alone should be recognised without much difficulty by means of the diplopia to which they give rise. It is a good plan to make a chart for one's self of every case observed, giving the patient a red glass in front of one eye, in order to make sure to which eye each image belongs.

So far we have seen under what conditions binocular diplopia is likely to occur, and how, by studying the relative positions occupied by the double images for different directions of fixation, it is possible to diagnose the nature of the defect in the muscular apparatus of the eye. As such lesions are often the result of cerebral changes, and may exist along with, precede, or follow other symptoms due to the same cause, it is evident that a correct diagnosis of the nature of the ocular paresis may be of great importance in enabling one to localize or even form some idea of the nature of the cerebral change. We have seen also under what circumstances conditions, which might give rise to diplopia, are not accompanied by that symptom, but there is one important aspect of the subject which yet remains to be considered. There may be abnormalities connected with the muscles of the eye which do not give rise to any manifest diplopia. This is the case when they are of such a nature, or are so slight as to be overcome by the increased impulse which a strong desire to maintain binocular vision brings about, that is, by the power of fusing the images of the two eyes. The defect may be either of a paretic nature, often the last remains of a paresis in the act of disappearing, or it may be of the nature of a muscular insufficiency, from some causes already referred to[1] as giving rise to this condition. In either case some degree of

[1] See Chap. I.

asthenopia is apt to result from the efforts made to overcome the difficulty. The method by which a defect of this nature can be detected is both simple and elegant; all that is necessary is to hold a prism of 4° or more with the angle directed upwards or downwards in front of one eye; this causes a vertical displacement of the images of the two eyes, which, as they cannot be brought to overlap each other, causes the patient to relinquish any effort he may previously have exerted to get rid of double vision, and to allow his eyes to assume a position which is more nearly one of equilibrium. In consequence of this he may observe, besides the vertical displacement, a lateral or torsional one, due to the paresis or insufficiency which is otherwise concealed. By examining the relative positions of the double images thus elicited for different positions of fixation, the nature of the muscular defect may be detected. The question of the range through which the power of fusion of double images into one exists, *i.e.*, of the breadth of fusion, is one of importance, as often affording a clue to the locality of the lesion, giving rise to some forms of paresis. Thus, if we find that when only a very trifling defect exists, causing but a slight separation in the double images, there is no tendency to overcome the diplopia; or if, for instance, in any case a prism whose optical effect counteracts that produced by the position of the eye only does so for one position, the diplopia appearing again as soon as the eyes are moved more or less slightly out of that position in the direction of action of the weakened muscle, there is evidence of a diminution, or it may be an abolition of the power of fusion. This would indicate, provided always that the case was not one of complete paralysis, and that there was good reason to infer that a normal degree of such power existed previously, that the cause of the paresis was central, *i.e.*, not only due to cerebral lesion, but caused by a lesion involving the more central regions of the brain. Cases sometimes occur, too, where, without the paralysis of any ocular muscle or muscles, diplopia suddenly comes on, owing to the loss, or paralysis of, the power of fusion. In such cases there has previously existed a divergent or convergent position of equilibrium of the visual axes, they indicate the

presence of some central change which is generally transitory, and possibly of a vasomotor or nemasthenic nature. Just as the use of a prism in the manner above indicated enables one to detect an existing insufficiency, what is called a latent or dynamic deviation, so in cases of paresis such a latent deviation becomes manifest, and influences the positions of the characteristic double images; for instance, the slight homonymous lateral displacement which should exist along with the vertical and torsional displacements in a case of paresis of the superior oblique may fail to make its appearance, or be replaced by crossed diplopia, when there is any considerable degree of latent divergence. Another circumstance which interferes with the arrangement of the double images which, in accordance with the physiological action of certain muscles, should characterize their paralysis, is the secondary contraction which often takes place, generally only after some time, in the antagonist. Thus the diplopia in a case of paralysis of the sixth nerve may not be confined to the side of the eye affected, but stretch to a greater or less distance to the other side as well, owing to a secondary contraction of the internus. These two conditions often add not inconsiderably to the difficulty of making a correct diagnosis.

Monocular Diplopia.—This may be only a variety of monocular polyopia—that is, it may be due to conditions which give rise to the formation of a number of separate retinal images, two of which irregular images are, however, more distinct and prominent than the rest; or the diplopia may be due to some cause which produces a mere doubling of the retinal image; or again, in some rare instances, it may result from an acquired faulty projection. The diplopia may exist even when the eye is correctly accommodated for the distance at which the object which appears doubled is situated, but the conditions giving rise to it are usually more likely to assert themselves when the eye is at the same time incorrectly focused for that distance. When suddenly complained of, it may either be due to some trauma, or the conditions causing it may have existed previously, and only be observed owing to some circumstance intervening which causes the attention to be directed to it. Of traumata likely to give rise to sudden monocular diplopia or polyopia may be mentioned the

piercing of the lens with a sharp instrument, the partial rupture of the ligament of the lens (zonule of Zinn), or paralysis of some portion of the ciliary muscle resulting in its irregular contraction. Or there may be dislocation of the lens, or separation of a portion of the peripheral attachment of the iris (indo-dialysis). Again, the trauma may be of the nature of an operation, the diplopia being the more immediate result of an extraction of a cataract, or following the operation of iridectomy for corneal opacity or synechiæ after iritis. Perhaps the most common causes of monocular polyopia are the changes sometimes occurring in the lens during the formation of cataract and the alterations of curvature of the cornea produced by inflammatory changes. It is also met with in cases of irregular astigmatism, and then the existence of the diplopia can be verified by ophthalmoscopic examination, a doubling of the vessels being particularly noticeable in this way.

The defect of monochromatic aberration common to all refracting surfaces which are not of such a form as to neutralize it, and therefore not what is called aplanatic, and which in optical instruments is practically to a great extent overcome by a centered system of surfaces of different curvature and different intervening distances, is to a great extent also rectified in the eye, partly owing to the dioptric arrangements, and partly, no doubt, also to the isolation of each percipient element of the retina. But there is a form of monochromatic aberration in the eye which does not exist in properly constructed optical instruments, which gives rise under certain conditions to polyopia. This aberration is due to the circumstance that the foci for rays passing through different portions (sectors) of the lens do not all coincide. When the eye is properly accommodated the want of coincidence of the foci is not sufficient to give rise to separate images, the only effect of the condition being that the retinal image of a point is larger than it would otherwise be. If, however, the retina be out of focus, the different images, instead of partially overlapping, are seen more or less distinctly as separate images. These images are crossed if the retina lies in front of the focal plane, and homonymous if it lies behind that plane; that is to say, that on

partially occluding the pupil the images of the opposite or of the same side disappear respectively. This kind of polyopia is very evident in cases of faulty refraction (ametropia), when a bright object for which the eye cannot be accommodated is fixed, and it is an exaggeration of this phenomenon which is the cause of the polyopia occurring in commencing cataract.

When not due to a trauma, the doubling of the image in one eye hitherto existing, though unobserved, may sometimes suddenly force itself upon the attention, either owing to increased retinal susceptibility to impressions, or to some cause having temporarily disabled the other eye, and when once discovered it may become more or less troublesome. In this way we sometimes find monocular existing along with binocular diplopia, the former being first brought to notice by the occurrence of the latter.

When diplopia exists in a case of dislocation of the lens the patient generally sees, for near objects of fixation at any rate, one distinct and one indistinct image, the more distinct one resulting from the focusing of those rays which pass through the portion of the lens opposite part of the pupil, while the indistinct one arises from the less complete convergence on the retina of the rays passing by the side of the lens. The relative positions of these two images differ in different cases according to the direction of dislocation owing to the prismatic effect of the edge of the lens. The diplopia in these cases of dislocated lens is one of the points which has to be taken into consideration in selecting the nature of optical correction best suited for improving vision; that is to say, in determining whether it is advisable to give concave glasses for the correction of the myopia produced by the displaced lens, or convex glasses for the correction of the hypermetropia resulting from the partial absence of the lens, as it sometimes happens that a glass otherwise suitable is of little use, owing to its rendering the diplopia more apparent and distressing; and this, again, is probably in no instance the case with both convex and concave glasses.

The question of the occurrence of diplopia owing to a doubling of the pupil has sometimes a practical importance in connexion with the operation of iridectomy. On looking at a needle

held vertically through two small holes some millimetres apart picrced horizontally in a card held immediately in front of the pupil, two images of the needle are seen if the eye be incorrectly accommodated for the distance at which it is held. This is the well-known experiment of Scheiner, and we may occasionally have somewhat similar conditions occurring in the eye as the result of disease or accident, the screen intervening between two pupillary apertures being either an opacity of the cornea or lens, or a portion of the iris tissue. It is seldom, however, that any of these conditions produce an appreciable diplopia, partly because there is in most cases at the same time a sufficiently accurate power of accommodation of the eye for different distances, but mainly owing to the intervening bridge which interrupts the rays of light being usually small, while the apertures which permit of their passage are not generally like mere pin holes, as in the experiment of Scheiner. It can be shown, as has been done by Schulek, that even when the eye is considerably out of focus only objects which are smaller than the intervening bridge give rise to double images, which accounts for the rarity of complaints of diplopia from patients in whom, from some cause or other, there is an opaque screen dividing the pupil into two parts. As a rule, the defect in vision produced in this way is not greater than to render the reading of very small type difficult or impossible.

I have alluded to one other possible cause of monocular diplopia. This I have never seen, but it has been described by several writers,—first, I believe, by Javal,—viz., diplopia due to acquired faulty projection. It is said that in some cases of strabismus the individual, when using one eye alone, may be conscious of seeing simultaneously two images—the one corresponding to the macula lutea, and the other to that part of the retina which has been for long associated with the macula of the other eye.

Monocular diplopia which does not admit of an explanation in some way in accordance with the manners above described, though the exact cause may be difficult to detect, probably does not exist, although some cases have been ascribed to purely cerebral causes.

CHAPTER III.

NIGHT-BLINDNESS AND DAY-BLINDNESS.

At one time it was supposed that that peculiar form of defect of vision which either only makes its appearance towards night, or becomes at any rate much more aggravated at that time, was, as it were, a kind of periodic weakness resembling ague in its nature. When, however, the attention of modern ophthalmologists was directed to the subject, night-blindness was soon found to be merely the expression of a defect in the light sense, a greater intensity of illumination being in general necessary in order that individuals in whom this defect exists should be capable of attaining their full visual acuity.

There is an unfortunate confusion as to the proper scientific term which should be accepted as synonymous with night-blindness. In the most recent English text-books the word nyctalopia is so used, whereas on the Continent hemeralopia has come into such general use that little or no attempt seems to have been made to change to the older, and from an etymological point of view more strictly correct name.[1] For this reason it is perhaps better to employ the English word night-blindness alone.

Physiologically there exists a condition in some respects allied to night-blindness. Thus, if we suddenly enter a darkened room after our eyes have been exposed to the full light of the day, we experience at first a much greater difficulty in recognising objects around us than we do after the lapse of some minutes spent in the comparative darkness. What is called an adaptation of the retina takes place gradually, and is the longer in

[1] We have the authority of Galen for deriving nyctalopia from νυξ and ἀλαὸς, and not from νυξ and ὤψ, and therefore using the expression to signify night-blindness, and not night vision or day-blindness, as is often done.

attaining its maximum the more intense has been the illumination to which the eyes were previously subjected. The time necessary for complete adaptation is also subject to individual differences depending on the state of the health, etc. If the eyes be subjected continually day after day to a more than usually intense light, such as is reflected from the surface of the sea or plains in tropical climates, or from chalk pits or snow, there is apt to be set up a condition which has been called *idiopathic night-blindness.*[1] This is more especially liable to occur if, along with the exposure to strong light, the individual is the subject of some weakness—malnutrition, anæmia, scurvy, etc. And although in much the greater number of cases of idiopathic night-blindness the primary exciting cause has been found to be referable to the action of strong light alone, or combined with such conditions as those mentioned, a certain proportion of cases appear to occur as a result of these debilitating circumstances independently of any abnormal conditions of illumination. Of this nature are the cases described as occurring occasionally in women shortly before confinement, and in some cases of cirrhosis of the liver and jaundice. The few cases which I have seen of idiopathic night-blindness, which is much more common in the southern countries of Europe and in the tropics, have been in sailors and soldiers who have returned from these regions. The affection apparently begins with a condition closely resembling that just described as physiological, but in which the period occupied by the adaptation of the retina is very much prolonged. Eventually the adaptation has not time to become complete before the individual is again subjected to the unfavourable conditions, and so a true night-blindness is more or less gradually acquired. The difficulties of vision come on during dusk, and in rooms illuminated by artificial light. Unless the illumination is pretty powerful, only objects directly illuminated by the source of light are seen distinctly, the others being more or

[1] The condition arises, then, from the illumination of the whole retina by strong irregularly reflected or scattered light; other symptoms, viz., a central scotoma, due to coagulation in the retina, are caused by the direct action of the light from strong sources of illumination, such as the sun or electric arc.

less indistinct according to the severity of the symptoms. The condition is one of anæsthesia or torpor of the retina, which requires an abnormally strong stimulus to awaken its physiological activity. Other symptoms are found to co-exist depending more or less on the same cause. Thus there is often, and to an extent varying in different cases, a restriction of the field of vision; there is, too, a certain amount of defect in the vision for blue, which leads to a tendency to confuse between shades of blue and green, a condition which is not characteristic of any form of true congenital colour-blindness. Alfred Gräfe has also described as complications defective accommodation and convergence, as well as a diminished tendency towards the fusion of double images produced by prisms. These are probably due to defective stimulation, to reflex or associated actions, owing to weakness of the afferent stimulus. They disappear along with the night-blindness. According to Reymond, the visual acuity of individuals suffering from idiopathic night-blindness does not begin to diminish until the strength of the illumination is reduced to a point which begins to tell on the normal acuity, but from this point there is a very much more rapid deterioration than in normal eyes as the illumination is further reduced. The indication for treatment is to withhold light so as to allow the retina time to recover itself, and in complying with this indication it is not necessary, as some have advised, to keep the patients in absolute darkness, but merely in considerably subdued light, either by the use of dark spectacles or residence in a darkened room, while at the same time attention should be directed, if necessary, to means calculated to improve the general health. Treatment carried out on these lines is always successful, and usually after a very short time. There appears, however, to be a decided tendency to relapse, which should be guarded against by a prolongation of the treatment after recovery. In a considerable proportion of cases of idiopathic night-blindness there is also a condition of xerosis of the conjunctiva due to the glare which gives rise to the defect of vision.

The form of night-blindness just described is accompanied by no

marked or constant ophthalmoscopic changes.[1] This is also mostly the case with the somewhat rare condition of congenital night-blindness, although it is highly probable that it is closely allied to the degenerative change of the retina known as retinitis pigmentosa; possibly the same disease occurring during intra-uterine life. Cases of congenital night-blindness have not, however, generally been found to be complicated by restriction of the field of vision, so that they may possibly be due to some defective development, a view which seems to be supported again by the fact that they remain stationary during life.

Much the most common causes of pathological night-blindness are conditions which, in most cases at least, produce visible ophthalmoscopic changes. Of such, again, retinitis pigmentosa is perhaps the most common, and certainly the most important. In this disease night-blindness is, indeed, usually the most prominent symptom, and the one which first attracts the attention of the patient or his friends; although complicated by other symptoms, chief of which is the almost constant diminution in the extent of the field of vision,[2] which is more and more marked the feebler the illumination. There often remains for a long time an excellent and sometimes a perfect acuity of central vision under a good illumination. The defect of peripheral vision renders the difficulties which patients suffering from this disease experience in comparative darkness all the more marked and distressing. They are able to go about perfectly well by themselves during the day-time, while they have often to be guided as soon as it is dusk. Unfortunately there is no cure for this disease, and therefore for the accompanying night-blindness—the changes occurring, though often slowly and even intermittently, being always in the direction of deterioration, and not infrequently leading to complete blindness. In some cases of retinitis pigmentosa, when the vision has become very defective, the night-blindness is not

[1] According to Poncet there is constriction of the arteries and dilatation of the veins of the retina, conditions which, if they exist at all, have certainly little to do with the characteristic symptoms.

[2] *Vide* Chap. vii.

marked, and sometimes even gives place to a kind of day-blindness, but I am not aware that this unusual symptom has ever been met with where there was good central vision. In most cases which I have seen for the first time in adults there has been night-blindness for long previously; occasionally, however, there is a distinct statement as to its having come on recently, so that although the disease usually begins in childhood, it certainly sometimes first makes its appearance in adult life. A not uncommon form of choroido-retinitis, in which there is also, as in retinitis pigmentosa, a deposition of the pigment of the hexagonal retinal cells in the more superficial layers of the retina, and which is usually if not always of syphilitic origin, is also associated with night-blindness. I have never, however, seen the night-blindness so pronounced in this inflammation as it is in the cases of true retinitis pigmentosa, which are sometimes confounded with it, and which are more of the nature of a degeneration or sclerosis than an inflammation in the limited sense of the term. Acute diffuse choroiditis, too, produces the most decided diminution of the light sense, and therefore night-blindness. Night-blindness is very evident, too, in some cases of detachment of the retina (idiopathic detachment), but not so much so either in the disseminated inflammations of the more peripheral parts of the choroid or the central choroiditis of old age. A certain degree of night-blindness, often sufficient to be rather inconvenient, is characteristic of the high degrees of myopia when associated with choroidal changes. Koenig has also described two interesting cases of defective development of the choroid, in which night-blindness was a marked symptom although the central vision was good.

When we come to inquire what is the pathological condition which gives rise to night-blindness, we must evidently look, in the first place, for some change which is common to all the affections in which it is met, and which at the same time is sufficient to explain the cases where the night-blindness is idiopathic and unaccompanied by alterations sufficiently grave to be visible with the ophthalmoscope. It is of course possible, though not perhaps so probable, that different conditions may give rise to the same symptom. Thus it

has been supposed, that as although most cases of idiopathic night-blindness have resulted from more or less prolonged subjection to diffuse light, still as undoubtedly the same defect may arise under such conditions as have been mentioned without the aid of that factor; the immediate cause may lie at one time in the retina, and at another in the nerve centres. Again, it has been ascribed to the limitation of the field of vision, and undoubtedly this limitation adds somewhat to the difficulties of night-blind patients, but it is more the condition of night-blindness itself which, by causing the more pronounced limitation of the field of vision in subdued light, gives rise to these exaggerated difficulties, than the defect of the peripheral vision, which brings about night-blindness. That this is the case is evident from the fact, that a good many cases of night-blindness are unassociated with constriction of the field of vision, and, besides, some forms of disease exhibit often a very pronounced restriction of the field, without producing at the same time the symptoms under consideration. Indeed, this is constantly the case when the restriction of the field is symptomatic of primary atrophy of the optic nerve fibres. Now, the experiments of Charpentier and others have demonstrated pretty conclusively that there are two distinct retinal end-organs, through the medium of which the transformation of physical states into the nervous stimuli leading to vision take place. One of these merely effects the transformation of energy, which gives rise to the perception of light, and is therefore, so to speak, the end-organ for the light sense; the other is capable of differentiating the impressions formed on it or on the former (or it may be the difference of the impressions formed on the two together), so as to lead eventually to a consciousness of the varying intensities and quality of the light rays falling on different parts of the retina, and thus give rise to the sensations of form and colour. The physiological night-blindness, so to speak, to which I have referred, shows that on exposure to light there is produced a certain degree of exhaustion of the light sense end-organ, so that a certain time has to elapse before it recovers itself sufficiently to be capable of its full delicacy. We have seen, too, that abnormal stimuli are capable of very much intensifying the

state of exhaustion, and consequently prolonging the period necessary for recovery. Thus it becomes a mere question of the balance between supply and demand, so that it is evident that an abnormal degree of exhaustion may be occasioned by either an excessive demand on the one hand, or defective supply on the other. There is, therefore, no occasion to relegate to different categories of causation the idiopathic night-blindness caused by strong light, and that resulting occasionally from debilitating disease or altered conditions of the blood, as both may evidently be due to exhaustion in the same end-organ. What that end-organ probably is we learn by a study of the more distinctly pathological conditions associated with the symptom of night-blindness. We find, then, that wherever there is absence or destruction of the retinal pigment cells, be it congenital or the result of inflammatory or degenerative changes, there night-blindness is a more or less marked symptom, and this independently as to whether other elements of the retina are affected or not. In the case of retinitis pigmentosa the symptoms are necessarily complicated, as apart from the disturbance of the retinal pigment there is the sclerosis of the vessels and connective tissue of the retina leading to its degeneration, and it is probably owing to this circumstance, as it is in connexion with this disease, that the symptom of night-blindness has been principally studied, that the cause of that symptom remained for so long obscure. Not only, however, have the more recent investigations into the physiology and pathology of the light sense clearly pointed to the hexagonal pigment cells as the end-organ for this factor in vision, but they indicate a method of examination by which in doubtful cases we may localize the cause of visual defects, and so determine whether they are of central or peripheral origin.

A degree of night-blindness is complained of by miners suffering from the condition of nystagmus, to which they are subject. This has not been referred to above, as it is certainly due to a different cause from that which gives rise to what may more correctly be called true night-blindness. It arises, in fact, merely owing to the increased nystagmus which is occasioned by the greater difficulty of seeing objects in the dark, which is common to all individuals.

Day-Blindness.

Although a considerable number of cases of day-blindness, in which vision in ordinary daylight is very imperfect, though of normal acuity, or at any rate much better in subdued light, are on record, occurring not only in isolated individuals, but endemically, yet this subject has not yet received a proper scientific investigation, and it is not known what is the exact nature of the disturbance which gives rise to this curious perversion of vision. It is said to arise in hot climates under much the same conditions which give rise to the much more frequent symptom of night-blindness, and to be some form of retinal fatigue. It is much more probably, however, allied to cases which indicate a degree of hyperæsthesia of the retina. There are few distinctly pathological conditions in which the symptom of day-blindness pure and simple is at all marked, so that on this account, too, it is difficult even to conjecture what may be the cause of idiopathic day-blindness. Cases of abnormal sensitiveness to ordinary daylight are indeed commonly enough observed, and such sensitiveness may be greatly increased by the habit of wearing darkened glasses, or keeping much in darkened rooms. How far such cases, in some of which there appears to be, along with the more prominent symptoms of hyperæsthesia, an actual improvement of vision in subdued light, are to be considered as analogous to day-blindness, does not appear very clear. Prisoners who have been kept for years in comparative darkness have been said to have become day-blind. Yet here again it is not easy to determine from the descriptions given how far the symptoms exhibited were those of hyperæsthesia alone. Albinos often have more difficulty in seeing in a bright than in a subdued light, owing to the excessive glare which the comparative absence of pigment in the iris and choroid permits of. The same difficulty, though for the most part much less marked, is seen when there is from any cause excessive mydriasis. Cases of cataract in which the opacity is central often see very much better in subdued light, owing to the dilatation of the pupil allowing rays to pass through more transparent portions of the lens. Such cases are consequently

improved for the time being by mydriatics. When there is central amblyopia due to diminished functional activity of the nerves supplying the central region of the retina, much the most common exciting cause of which is the toxic effect of tobacco, patients often state that there is an improvement in their vision when the surrounding light is subdued. In cases which I have specially examined with reference to this point, I have not noticed any actual increase in visual acuity when the light has been diminished, but rather the reverse, so that it is probable that such a condition is merely associated with a greater sensibility to glare, which might cause what is so often complained of, the appearance of objects being less distinct in a bright than in a subdued light. The only cases which, so far as I have observed, present symptoms resembling true day-blindness are some cases of reflex or hysterical amblyopia, in which, if one can trust the statements of the patients, there seems to be a more or less distinct improvement of vision in subdued light. In one case, in which there was a marked restriction of the field when tested by ordinary daylight, I found the peripheral vision normal in extent in a darkened room. But these cases more frequently present the symptoms of anæsthesia of the retina, and evidently from a different cause from that which has been explained as giving rise to night-blindness. The constant irritation of sensory nerves in different parts of the body, which produces in them the greater or less degree of amblyopia, to account for which no ophthalmoscopic changes can be found, does so by unconsciously alienating the attention from the impressions produced on the visual centres when images are formed on the retina, just as the absorption of the attention by the impressions of one or more senses may make us unconscious of the stimuli produced on the centres of other senses. And that such is the case is shown not only by the fact that reflex amaurosis and amblyopia may be suddenly cured when the source of primary irritation has been removed, but also that in these cases the pupil is not generally inactive, as it is, more or less, when the cause of the blindness is peripheral to its reflex centre. Such cases suggest an explanation of day-blindness. We have said that this condition is allied to hyper-

æsthesia of the retina, and it is not unlikely that this hyperæsthesia, in which this action of light produees a kind of irritation of sensory nerves either in the retina or somewhere in connexion with the nervous apparatus of vision, and whieh appears to be engendered by the subjeetion of the eyes for longer or shorter periods to brilliant light, may give rise to mueh the same kind of blindness as that to which we eommonly give the name of reflex amblyopia, with this differenee only, that as the original souree of irritation proeeeds from the visual apparatus, and is eaused by light itself, it would be less, and therefore admit of better vision when the illumination was diminished to an extent which rendered the irritation less perceptible. That is, there would be likely, in eases where the hyperæsthesia existed without so much pain as to give rise to the spasmodie elosing of the eyes, to be an improvement of vision in subdued light, or, in other words, day-blindness.

CHAPTER IV.

METAMORPHOPSIA.

We may distinguish between two forms of metamorphopsia; complete metamorphopsia, that is complete in the sense of existing for all surrounding objects independently of the part of the retina on which their images fall, and partial metamorphopsia, where the symptom of distortion is only noticeable in the case of objects whose images are formed on definite portions of the retina. In either case the change of shape may be regular or irregular, and characterized by an apparent enlargement of objects (macropsia), or be of such a nature as to produce an apparent diminution in their size (micropsia). Of partial metamorphopsia the most distressing is that which is symptomatic of certain changes in the region of the macula lutea, as apart from the distortion to which these give rise there is generally at the same time more or less interference with visual acuity.

Our judgment of the shape of objects seen, depends in the first place on the configuration of their retinal images; it is therefore the more in accordance with their actual shape the more regular is the refraction of the rays of light through the dioptic media of the eye, and the more accurately the rays are focused on the retina. For the correct appreciation of form, it is further necessary that the percipient elements of the retina should be grouped according to their normal arrangement in the focal surface of the eye, and should all be capable of excitation to some extent at least. There is also, of course, the psychical element, which, in this respect, as well as always when there is a question of the interpretation of the impressions of the senses, has to be taken into account. Various illusions and delusions occur as to form and dimensions, as well as

in connexion with other impressions and other special senses. These constitute, however, for the most part at least, a chapter in the physiology and pathology of the central nervous system, and do not call for consideration as symptoms of disorders of the eye.

Of the size of objects, again, we judge partly from the size of their retinal images, but as this, besides depending on their actual size, depends also on their distance from the eyes, the appreciation of size is more correct the more accurately the distance is gauged. In comparing the sizes of two or more objects at the same distance from the eyes, we are probably guided by the appreciation of their retinal images alone if they are small. When of any considerable size, however, the method of comparison is more complicated, and depends also upon the knowledge gained by the muscular sense on running the eyes along over the different points of the objects.

There are, therefore, evidently many different possible causes of metamorphopsia, as we may receive erroneous conceptions of size and configuration owing to the existence of abnormal conditions altering the character of the impressions gained in one or more of the above-mentioned manners.

In cases where there has been any accident to the cornea, or where its tissues have been altered by inflammation or other pathological processes, it is apt to lose its normal curvature, and thus the images which are formed on the retina become more or less distorted, and consequently less distinct. This, which is called corneal astigmatism, may be regular, that is, due to an altered curvature having taken place, and therefore producing a distortion in one direction mainly, or irregular, owing to circumscribed alterations in curvature which have been produced in different directions and to different extents, and therefore give rise to irregularly distorted images. While the regular changes can, to a great extent, be counteracted in their effects by suitable optical means, the irregular rarely admit of much improvement. Changes in the curvature and refractive power of different parts of the lens may in the same way give rise to metamorphopsia owing to lenticular astigmatism. Irregular corneal astigmatism is usually pathological,

irregular lenticular astigmatism often physiological, that is, not at any rate the result of disease.

Occasionally we find in cases of nebulæ of the cornea irregular distortion complained of. Such patients sometimes, though less frequently than one would imagine, see parallel lines bulged outwards or inwards, and often at the same time irregular outlines presented by circular or curved lines. This phenomenon is, no doubt, partly due to the alteration in curvature of those parts of the cornea which have remained clear, or have cleared up after the inflammation which has led to the nebulæ or circumscribed opacities has subsided; but it is also not unlikely that in cases of dense central nebulæ at least, and when the retina lies out of focus, it is caused merely by the circumstance that the rays which should pass through the centered portions of the curves of the cornea and lens are excluded, while the more peripheral alone go to form the image.

Whilst the metamorphopsia to which lenticular as well as corneal irregular astigmatism gives rise seldom fails to attract the attention, especially if the individual takes notice of the impressions he receives, it is usually otherwise with that which is caused by regular astigmatism, when occurring congenitally at least. Although it is difficult to say in what manner many astigmatic individuals see,[1] because the form of the retinal image, which is for most objects always indistinct, depends on the state of accommodation of the eye, still it is clear that there must often be not only an enlargement of the image owing to the circles of diffusion, but also a

[1] There are many possibilities: In the cases of astigmatism where it is possible the eye may be accommodated for the position of least diffusion, between the foci of rays through the principal meridians; there may be more or less disregard acquired for the areas of diffusion; there may be a rapid unconscious change of accommodation permitting a cerebral elaboration of the successive distinct portions of the image, while the other portions are disregarded. There are many reasons, however, for supposing that in most cases, at anyrate, the eye is focused for the rays refracted in the plane of one of the chief meridians, in which case any object, except lines at right angles to that plane, must necessarily present a distorted retinal image. The further discussion of this point does not call for consideration in connexion with the subject of this paper.

distortion of it, as the enlargement thus produced is greater in one direction than another. It is evident, then, that many must see circles in the shape of ellipses and squares as oblong figures, yet it is rare to hear a complaint made of such a distortion. And, indeed, except when the differences in the shape of the indistinct image of a bright object, such as the moon when seen under different states of accommodation, is observed, it is difficult to see how one should become conscious of the abnormality. Very often, indeed, even although the vision is much improved by correction with cylindrical glasses, the difference in the shape of bodies with and without correction is hardly apparent, or at any rate by no means so marked as the action of cylindrical glasses on those who are not astigmatic would lead one to suppose. In cases where the astigmatism exists in one eye alone, or is more marked in the one eye, there is a consciousness of the different apparent shapes of objects in the two eyes when the individual has directed attention to the impressions received on either eye separately, but even then there is often, and unless the anisometropia is considerable, no consciousness of distortion, owing to fusion of the two images. On the other hand, the more or less regular astigmatism which results often from the operation for the extraction of cataract, being produced, as is frequently the case in an individual who has had no marked degree of astigmatism previously, gives rise to a distinctly noticeable metamorphopsia owing to the comparison which he is able to make between the images of objects of known shape after operation and those previously experienced. A curious form of metamorphopsia is experienced by some myopic individuals when wearing the spectacles which correct the error in their refraction. They see lines on a flat surface at right angles to the horizontal visual plane curved towards them. Sometimes this is so distinct as to cause a good deal of annoyance. This, which is due to the spherical aberration of the glasses, is not complained of to anything like the extent which one would expect, owing no doubt to the want of habit in most individuals of observing at all closely the visual impressions which they receive. The same kind of distortion is much more frequently noticed by intelligent individuals when

making use of the glasses necessary after cataract extraction; but in all cases is not long in disappearing or ceasing to cause inconvenience. Myopes, too, often complain of everything appearing smaller when they first begin to wear the necessary glasses. There are two reasons for this:—In the first place, all objects beyond their far point have always, owing to circles of diffusion, appeared to them rather larger than they do when their images are more correctly formed on the retina; and, in the second place, objects within their far point of distinct vision form somewhat smaller images on the retina when looked at through the correcting spectacles than without. The corresponding increase in the size of the retinal images when hypermetropes use glasses instead of their accommodation is not so often complained of, because not so disagreeable. In the case of aphakia the apparent size of objects, *i.e.*, whether they appear larger or smaller than was the case previously, depends greatly on the glass used—the weaker the correcting glass, and therefore the further away from the eye it has to be placed in order to focus the rays from any object on the retina, the greater is the size of the retinal image obtained.

We have said that the apparent size of an object, besides depending on the size of its retinal image, depends also on the distance at which it is taken to be at from the eye, and the more or less complete knowledge, gained by experience, of the approximate actual size. If we hold up a finger in front of one eye it is found to cover a portion of a house or tree beyond it, the size of which appears nevertheless very much greater than the finger. An estimate of distance, again, is often very much facilitated by the knowledge of the actual size of the object, but where this factor does not come into play, it is arrived at consciously or unconsciously by a variety of circumstances influencing both the monocular and binocular impressions received. Thus monocularly there are the colour, shade, existence of shadow, and under certain conditions the parallax, and in the case of near objects the degree of accommodation which is required to obtain a distinct image, which singly or combined afford data on which our judgment may be based. And where this is possible we are greatly guided, no

doubt, by the differences in shape of the retinal images received by approaching the eye to the object through a certain range, of the extent of which we form an unconscious appreciation. Binocularly, there is in addition a consciousness of the degree of convergence of the optic axes which is required for fixation with both eyes of points at definite distances, when the relation between such degree and the actual distances has been more or less unconsciously acquired; there is also the stereoscopic effect resulting from the cerebral elaboration and fusion of dissimilar images of objects in the two eyes. A familiar example of error in judgment as to size, is that of a fly crossing in front of the eyes and, being unconsciously projected by the mind against a considerably more distant plane, appearing as a large bird. Such a pronounced misjudgment only occurs when neither the attention nor the visual axes are directed on the near object. But even when both eyes and attention are fixed on a particular object it may, though at a constant distance and subtending always the same visual angle, appear of different sizes owing to the different distances to which the mind may project it in space. It is owing in great measure to this circumstance that the moon, for instance, on the horizon appears larger than when higher in the heavens—our mental projection of the vault of the heavens being not a hemisphere, but only a portion of one. The apparent size of the sun and moon is very different to different individuals, but is always greater than can be accounted for by the size of their retinal images projected on to a plane near the eyes.

Defects of accommodation give rise to alterations in apparent size owing to the error in the judgment of the distance of objects within the ordinary range of accommodation which results from them; thus, when all at once there is a paresis of accommodation, and a greater effort has to be made in order to focus a near object distinctly, or to attempt to focus it distinctly, this gives rise to an impression of greater proximity of the object, and as its image on the retina is of course of the same size as it would be were its distance more correctly estimated, it appears smaller. The micropsia thus occasioned is often very marked, and is met with both in cases

where the paresis is the result of a direct or reflex interference with the function of the branches of the third nerve which supply the ciliary muscle, which may occur idiopathically or be due to the action of a mydriatic. Conversely a spasm of accommodation, from whatever cause, sometimes gives rise to macropsia, as the consciousness of an abnormally feeble effort to accommodate the eye for a near object causes the object to appear more distant, and therefore bigger than it would otherwise be estimated. Macropsia has thus been observed after the use of such myotics as pilocarpine and eserine. Accommodative micropsia is most marked the nearer the object lies to the eye; accommodative macropsia, on the other hand, the further it is removed from the eye.

But besides metamorphopsia due to defects in accommodation, there are also similar misjudgments as to size, caused by abnormally impeded or facilitated movements of convergence of the optic axes, or any optical conditions which permit of binocular fixation with the axes of vision directed so as to meet either nearer or further off than the object looked at. Thus prisms with the bases inwards in front of each eye appear to magnify owing to the axes of vision meeting further off than the objects looked at, giving rise to an impression of greater distance, while prisms with the bases outwards in front of each eye appear to diminish, as under these circumstances the axes of vision cross in front of the objects fixed, which appear nearer, and consequently smaller, than would be the case without the prisms. Operations on the muscles of the eyes are sometimes, if there has been previously binocular vision, followed by similar apparent alteration in the size of near objects.

So far the changes in shape of objects that have been considered have been such as were due to optical causes or others not symptomatic of any very grave disorders of vision. It is otherwise when the cause of the metamorphopsia lies in the retina, when owing to some alteration in the relative positions of the percipient elements an alteration takes place in the external projection of the luminous impressions which they receive. The retinal elements may be displaced forwards by an exudation of fluid,

which according as it is serous or fibrinous, and general or localized in patches, and as to whether it involves the more peripheral or more central portions of the retina, is accompanied by subjective symptoms, varying both in their nature and severity. The exudation may be in the retina itself, or may give rise to swelling of the retina, so that a smaller number of its elements come to occupy the same superficial area as before; or, in other words, the same number of elements are spread over a larger space than under normal circumstances. The image of any object falling on such an area produces a stimulation of a smaller number of retinal elements than it would otherwise do; but the projection being unaltered, this is not compensated for, and the object appears diminished. On the other hand, the shrinking resulting from the contraction of such a fibrinous exudation may lead to a greater approximation of the elements in the area involved, so that the same number of elements are spread over a smaller space than they originally occupied. It will be readily understood how, in a similar manner to that described in the case of a separation of the elements, such approximation must give rise to macropsia. This swelling or contraction may take place regularly or irregularly in all directions, or more in one or more particular directions than in others, consequently the metamorphopsia to which it gives rise may exhibit a corresponding degree of regularity or irregularity. Thus although the distortions in cases of disease of the macula usually occur more or less symmetrically round the point of fixation, such irregular changes—as, for instance, rupture of the choroid, which, owing to the greater firmness of attachment of choroid and retina to the sclerotic at the posterior pole of the eye, almost invariably takes place between the macula and optic papilla—are accompanied by a correspondingly irregular metamorphopsia. The apparent distortion of objects produced by detachment of the retina is usually so marked as to spontaneously attract the attention of the patient. It is not only irregular, but frequently inconstant, changing like the images reflected from the surface of disturbed water. Metamorphopsia from this cause is often, too, distinguished by the distorted objects being bordered by colours.

In diffuse choroido-retinitis, a form of inflammation which is often of syphilitic origin, there is very generally a pretty regular metamorphopsia which usually takes the form of micropsia, sometimes, however, of macropsia. The micropsia is frequently very marked, particularly if the affection is confined to one eye. It usually only asserts itself after the inflammation has existed for some time, and often remains for long after it has been recovered from. Foerster, who was the first to explain the cause of retinal metamorphopsia, pointed out that the micropsia increases as the distance of the object from the eye becomes greater, thus showing that it is not connected with any coexisting defect in accommodation. I have not seen macropsia caused by this disease, but it is described by Ole Bull in his recent work on *The Ophthalmoscope and Lues;* and there seems no reason why it should not sometimes be a later manifestation of the same lesion which so commonly produces the opposite distortion. The condition of macropsia following on micropsia always, indeed, suggests the choroid as the site of the inflammatory change. Where the lesion has been from the first undoubtedly retinal, I have seen the micropsia to which it gave rise remain permanent. When the metamorphopsia does not exist over a large area of the field, or involve the centre, it is sometimes only to be made out by a careful subjective examination. This may be done by causing the patient to fix a spot in the middle of a number of parallel lines a few millimetres apart on a piece of paper, which is held so that the lines are alternately vertical and horizontal, and asking him whether they appear all to run parallel, or bend outwards or inwards, at any particular place.

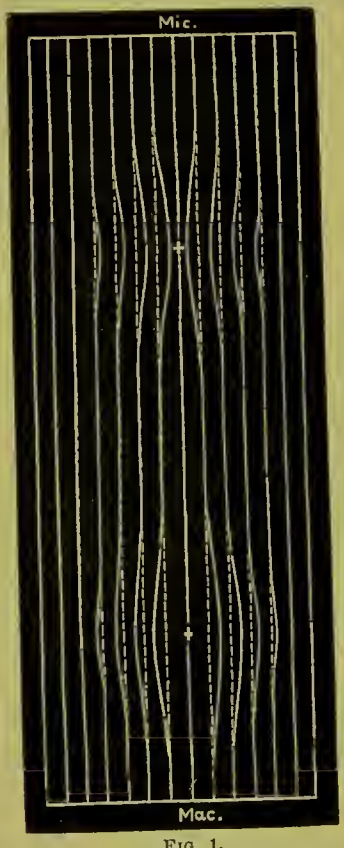


Fig. 1.

In this way we may detect not only the existence of a localized metamorphopsia, but also whether we have most probably to do with a recent exudation causing a distension, or an old exudation which has led to a contraction of the retina in that area. In the first case the lines will appear to bend inwards, the projection of the retinal images in that position being smaller; in the second case they will appear to bend outwards, the projected images being bigger than normal. Figure 1 shows the distortion indicative of micropsia and macropsia respectively. In cases of progressive myopia there is frequently metamorphopsia owing to the changes which take place in the region of the macula lutea. As I have found the distortion in this condition to take the form of macropsia, it seems to me probable that sometimes, at any rate, these macular changes are due to the contraction of exudations in the subjacent choroid which involves the external layers of the retina, more than to actual stretching of the retina. Possibly the distortion might be due, however, to micropsia occasioned by stretching of the surrounding and less firmly adherent parts of the retina. The senile change which takes place in the vitreous membrane of the choroid close to, or immediately behind, the macula, gives rise to an irregular metamorphopsia, which may be detected in the manner described. Retinal hæmorrhages when near the macula often leave behind them a similar condition of metamorphopsia. Sometimes such changes are not easily seen with the ophthalmoscope, in which case the presence of metamorphopsia is of value in directing one to a correct diagnosis.

CHAPTER V.

PHOTOPSIA, CHROMATOPSIA, MYODESOPSIA, Etc.

A SENSATION of light may arise even when there is no objective cause to account for it—that is, even when no undulations of the ether, such as are capable of giving rise to luminous impressions, find their way to the retina. A purely subjective sensation of this kind may be the result of a mechanical, electrical, or chemical stimulation of the nerve fibres in the retina or optic nerve, or of the centres of vision. The light so seen may appear coloured or uncoloured, according to the site and nature of the stimulation. Further, under certain circumstances, coloured luminous impressions may be received when the objective source is of a nature which should only give rise normally to uncoloured light sensations. This may be due either to causes which separate the compound white light into its constituent rays, or to such as give rise to a selection of some rays, and at the same time a greater or less absorption of others.

Many are familiar, probably, with the brilliant colours which can be elicited in the dark by light continued pressure on the eyeball. The pressure generally first produces a colourless, ill-defined cloud, which gradually extends in all directions towards the periphery of the field of vision, and is followed by colours, blue, red, and green, etc., which, beginning at the centre, spread outwards in the same manner as gradually widening rings towards the periphery. On continuance of the pressure, these colours become less vivid and less regularly disposed, until the field, after some time, becomes entirely dark. On relief of the pressure, there are again produced light sensations, which are, however, much more irregular and not so variedly coloured. Direct pressure over a portion of the eye

behind its equator also produces in the dark a so-called phosphene or sensation of light, which is referred to the opposite side of the field of vision. If a similar pressure be made in the light instead of in the dark, it is a darkish spot which is seen, the retina being rendered apparently by such mechanical irritation less responsive to stimulation by light. The slight stretching which the retina is subjected to by the action of the ciliary muscle also gives rise to faint phosphenes, which may be experienced by suddenly relaxing the accommodation in the dark. Phosphenes also occur in forced movements of the eye. These appear to be most marked on turning the eye upwards, and are probably due more to intermittent pressure of the oculo-motor muscles in the eyeball than to stretching of the optic nerve by the movement. Electrical stimulation also produces light sensations, mostly coloured. According to Schliephake, closing of a current ascending the optic nerve and opening of one descending produces a momentary bluish-violet illumination of the whole field of vision. Others describe also a yellowish tinge when the galvanic current descends the nerve. Of chemical irritants, that which has been most studied is santonine. This substance produces, according to Woinow, first the sensation of a violet, and then of a greenish-yellow colour, which is sufficiently marked to cause objects looked at to appear yellow. Its further action seems to increase the visual acuity without giving rise at the same time to coloured vision. Woinow explains the action of santonine by assuming that it first produces a stimulation of the violet-perceiving elements of the retina, which is followed by an exhaustion of these elements, while in the case of an increased visual acuity alone all the colour elements are equally excited This explanation is, however, based on the prevalent theory of colour vision, which is in many respects very unsatisfactory.

The presence or absence of colour in the phosphenes, due to stretching of the retina from any cause, may be of importance as affording some indication of the portion stretched apart from the locality of the field to which they are projected. The most peripheral portions of the retina are either totally colour-blind, or require a very much greater stimulation to give rise to

colour impressions than more central portions. Consequently, when they alone are mechanically stimulated, the phosphenes are colourless. As a general rule, indeed, colourless phosphenes are due to stretching of the anterior part of the retina. Such colourless and indefinite phosphenes are complained of, too, where there is slight eyelitis. They take the form of more or less incomplete circles, and, as they are not constant, are in all probability brought about by movements of the ciliary muscle, which cause dragging of or pressure on the hypersensitive anterior part of the retina.

Mayerhausen has lately studied a form of photopsia depending on pressure caused by over-dilated vessels in the retina. The sensation to which pressure of this nature gives rise is that of faint irregularly defined streaks of light disposed in a radial direction round the point of fixation. The length of these streaks is variable. When long they branch dichotomously, and gradually fade away at the ends, never terminating abruptly. They disappear when the eyes are closed. The vaso-motor disturbances giving rise to this kind of subjective light sensation occur in constitutionally nervous individuals suffering from more or less neurasthenia, and the vessels through whose over-distension the symptom is caused are probably the outer network of capillaries which are mostly situated in the inner nuclear layer of the retina. Mayerhausen was able to study more closely than had previously been done the nature of this form of photopsia as under certain conditions, viz., violent exertion following prolonged use of the eyes, and at a time when the nervous system was exhausted by fasting, he was conscious of the sensation himself, which can therefore only be looked upon as pathological when arising independently of such abnormal conditions. Sudden loss of vision, due to embolism of the central artery of the retina, very often occurs at night and escapes observation, but it is said that in cases where its occurrence is noticed by the patient there is at the same time a consciousness of subjective light sensations. Inflammatory conditions of the retina or nerve do not appear, as a rule, to cause photopsia, thus it is seldom that in optic neuritis any complaint

of this kind is made. In glaucoma, however, besides the coloured halos round lights to be afterwards more particularly referred to, there are frequently subjective colour and light sensations which seem to be due to pressure. In cases of diffuse retinitis or choroido-retinitis, where there are generally to be found areas in which the retinal functions are defective, there is often at the same time a sensation of coloured and generally flickering spots of different shapes and sizes complained of. These are always referred to the portions of the field which may be found on examination to correspond to the areas of the retina in which the visual defects are most marked, and are the result of irritation or dragging on the retinal elements. The subjective light and colour sensations met with in this disease often continue for long after the inflammation has quite subsided. They are most evident, as a rule, after any prolonged exposure of the eye to strong light, or after anything causing fatigue, either of the retina or body generally. Conversely they tend to become less marked or to disappear when the eye is rested, and therefore at night, in which respect photopsiæ of retinal origin differ from those caused by irritation of the visual centres, which are often most distressing during the night and at times when the eyes are not subjected to stimulation from objective sources. Retinal photopsia often continues for long after there is complete blindness, and is sometimes, though rarely, even so severe and distressing as to call for enucleation of the eye. Plateau, who was blind for forty years, had constantly subjective light sensations proceeding at different times from irritations of different portions of the retina, but always projected in accordance with the direction of the movements of the eyes. Notwithstanding the degenerative change which takes place in the retina, and is characteristic of retinitis pigmentosa, it is only very rarely that patients suffering from this disease are troubled with light or colour sensations. The effect of dragging or stretching in producing photopsia and chromotopsia is, however, most marked in many cases of detachment of the retina where such subjective sensations usually accompany the occurrence of the detachment, and are often complained of from time to time as the separation

becomes more and more extensive. The colours mostly seen are red and blue, and mostly at the beginning of the affection, the wavy distorted outline of objects being often then bordered by these colours. In cases of sclero-choroiditis anterior, especially when the affection proceeds to the formation of staphylomata, subjective sensations of light and colour are very frequent. These, too, are probably mostly due to abnormal stretching, though they may possibly, to some extent, arise from inflammatory irritation as well.

Closely allied to cases of photopsia due to pressure on, or stretching of, the retinal elements, are those which appear to originate from circulatory changes, often of the nature of vaso-motor disturbances,—recurrent paralyses of the vaso-motor nerves of the retina. In some cases, too, of hyperæsthesia of the retina, complaints of coloured vision are made. This kind of chromotopsia is evidently the result of the response to stimuli which would otherwise, on account of their feebleness, be disregarded, but which are observed owing to the irritable condition in the retina itself, or in the more central portions of the visual apparatus, just as hyperæsthetic individuals may be affected with pains for which little or no objective cause can be detected.

A curious form of subjective colour sensation has recently received a good deal of attention. It has mostly been met with in aphakia after cataract extraction, and consists in a more or less constant sensation of everything looked at being coloured a vivid blood-red. There is no veiling of the objects, but merely a marked red coloration. The condition to which the name erythropsia is generally given has not yet received a satisfactory explanation: it has been ascribed to the dazzling caused by light rays passing through the coloboma in the iris, or to that along with some fatigue of the retina. As it is met with under other conditions than aphakia and coloboma of the iris, and is by no means a common occurrence, it is impossible that these should in themselves be of paramount influence in the production of the erythropsia. Hilbert, for instance, has described the sensation as it has several times occurred to him. Though with otherwise normal

eyesight, he has had several attacks of erythropsia, which have always come on after sleepless nights, and have disappeared after a good meal. When it occurs after cataract extraction, it seems usually not to come on until some months after the operation, and in cases, too, where everything has gone well, and good vision has been obtained. Thus Hirschler has described how, five months after having been operated on for cataract, he was troubled with this red vision, which lasted for two months and then disappeared. The only case which has come under my own observation after cataract extraction occurred one month after the operation, perfectly suddenly, and continued for several months. In this case, a healthy woman of 50, who obtained excellent vision ($\frac{20}{40}$), and from whose iris I had, as I am in the habit of doing, only removed a small portion, so that the resulting coloboma was small, and entirely covered by the lid, there was no evidence of any connexion between general fatigue and the symptom of red vision, which, though not constant, came on every day, and as often after a meal as before it. She herself ascribed it to glare, although there appeared to be no unusual circumstances of exposure to light connected with the case. The other eye was affected with immature cataract. Ophthalmoscopically there was nothing to account for it, and the symptom did not interfere with reading. Another case occurred in an extremely nervous lady, and came on after an exhausting mountain climb in Switzerland, the eyes having been subjected at the same time to a prolonged reflection of light from snow-peaks and glaciers. In this case there certainly was a coloboma in the only eye in which there was vision, the other having been lost by atrophy after a severe fall on the head, which had probably therefore caused fracture through the orbital foramen. The view entertained by Benson, Purtscher, and others who have met with cases of erythropsia, viz., that it is due to hyperæsthesia of the retina, affords certainly the most probable explanation, though whether the condition of the retina is favoured by aphakia and the presence of a coloboma in the iris, or is brought about in some other way, is not clear. The following experiment, described by Chevreuil, shows the influence of strong

light shining into the eye in producing a somewhat similar condition to erythropsia. Chevreuil allowed the sun to shine into his right eye while the left was closed. After two minutes' insolation, black objects on the table appeared to the right eye red, and white, green, whilst with the left they were seen in their proper colours. This he gave as the explanation, too, of the historical "blood spots" which came before the eyes of Henri IV. and the Duc d'Alençon whilst playing chess in the open air a few days before the massacre of St Bartholomew.

The influence of fatigue or hyperæsthesia of the retina in giving rise to coloured vision is therefore undoubted. Some poisons which cause nervous fatigue occasionally also give rise to chromatopsia. Thus red and green vision has been met with by Bruce and others in cases of poisoning by bisulphide of carbon. Whether such poisons act by irritating the retina, or the centres which have to do with the perception of colour, is not certain. The latter seems the most probable, as not only are there conditions met with which point to the existence of such centres, such as hemianopia for colour alone, but the poisons produce at the same time other cerebral symptoms as well. Indeed, it is just possible that the cause of erythropsia in aphakia may be a central irritation, unconnected altogether with operations for cataract, though associated, perhaps, with the conditions which have given rise to the loss of transparence in the lens.

Coloured vision may be produced again by the actual presence of coloured substances in the eye. Thus, when blood is effused into the vitreous, there is for a short time the sensation of a red veil covering the indistinctly seen objects. In cases, too, of hæmorrhage into the retina a red veiling is often seen which afterwards gives place to a yellowish or greenish discoloration of objects. In detached retina there is frequently, besides the colour and light sensations, due to stretching of the retina, an appearance of a yellowish or greenish veil complained of, sometimes though, the appearance is that of a more or less distinctly blue veiling. This latter Leber supposes may be due to contrast from the yellow subretinal fluid. Many authors have described a condition of yellow vision

or Xanthopsia in jaundice. It is certainly not a common occurrence, however, and when it does occur is probably due to a yellow discoloration of the dioptric media, which for some reason or other is not often occasioned in jaundice. Here again, however, the pathology is not clear, although the explanation given seems the most probable one.

A common cause of coloured vision, owing to a change produced on the compound rays of white light as they enter the eye, is afforded by those states of the cornea which give rise to diffraction. This can only in so far be looked upon as a subjective sensation, in that the physical conditions, as far as the light is concerned, are such as would not under normal circumstances give rise to the sensation of colour. In looking at the street lamps, for instance, through the glass of a carriage window, which is covered with the closely packed minute particles of moisture which arise from the condensation on it of the vapour within, one frequently notices the lights surrounded by coloured halos. The same appearance may sometimes be observed without the intervention of the glass, and is then usually pathological and due to a similar arrangement of intransparent particles in the cornea. This symptom of seeing halos round lights is one of the commonest and most important indications of the early stage of glaucoma. The slight attacks of increased tension which only occur at intervals are accompanied by this symptom. When a flame of a candle or a gas jet is looked at, it is surrounded by a circle or halo of colours, which is separated from it by an uncoloured space. This uncoloured space is light where it immediately surrounds the flame, and dark towards the coloured rings, and the space as well as the coloured rings are both broader the further the light is from the eye. The most external colour in the halo is always red, the most internal bluish or bluish-green, while at different parts of the circle there are brighter spots, so that the halo is not absolutely regular. The colours are seen with varying degrees of intensity in different cases, and are even sometimes, when very distinct, surrounded by a second halo, like a second rainbow, with this distinction, however, that the arrangement of colours in the second halo, which is much less

distinct than the first, is similar to the first, and not reversed as in the ease in the secondary rainbow. This is a proof that the phenomenon is due to interference or diffraction. A further proof of this, which was pointed out long ago by Donders, is that on covering the lower half of the pupil, the outer and upper and the lower and inner quadrants of the halo disappear. The halo, too, always remains stationary whether the light be fixed by the eye or not, or whatever be the state of aeeommodation of the eye. The eause of the distinetness of this appearance in glaueoma is probably owing to the haziness of the cornea and the simultaneous dilatation of the pupil. It occurs in non-inflammatory as well as in inflammatory glaueoma, so that it is in all probability due to the optical conditions which are introduced by disturbances in the lymph eirculation within the cornea, although it has not yet, I think, been quite definitely proved that other of the dioptrie media, more especially the lens, do not participate in the haziness. Although a frequent symptom in glaucoma, such eoloured halos are by no means pathognomonie of that disease. They may be seen in all cascs where the eornea assumes from any cause a diffuse cloudiness. Even the secretion, whieh in some eases of conjunetivitis gathers across the eornea, may give rise to them. When spontaneously complained of, and espeeially when there is no appearance in the cornea at the time of examination to aecount for the halo, and when, too, it does not disappear on rubbing the eyes, its existence is strongly suggestive of glaucoma.

One form of subjective light sensation, viz., that which is in all probability of cerebral origin, still remains to be eonsidered. One of the most eommon forms of this kind of photopsia is that asso-eiated with hemierania. The symptoms of this peculiar affection are probably now well known to most medical men. It may begin in different ways, and last for a longer or shorter time, usually after prolonged bodily or mental fatigue, or at a time when there is more or less nervous exhaustion from want of food or sleep, it suddenly makes its appearance as a dark spot to one side of the field of vision in both eyes. The dark area slowly increases in size, and after some time becomes bordered by scintillating and often

coloured zig-zag margins of greater or less intensity. The configuration of these margins of light often resembles the angular wavy outline of a fortification, and for this reason Airy gave to the affection, from which he himself suffered, the name of Teichopsia. The duration of the whole visual disturbance is generally rather less than half an hour. The appearances fade away from the centre towards the periphery, and are most frequently followed by severe headache, often accompanied by sickness, which lasts for several hours. The central nature of the subjective sensations just described is pretty definitely established by the fact that they occur, and, indeed, this is most frequently the case, in true hemianopic form. This could only be the case when the temporary disturbance, whether vaso-motor or of whatever other nature, was situated centrally with respect to the chiasma, unless possibly a vaso-motor disturbance took place simultaneously in symmetrical halves of both retinæ. The latter possibility is unlikely, partly because it would not be in accordance with the anatomical arrangement, so far as is known, of the nerve fibres which supply the retinal vessels, but more especially because no such disturbance can be detected by ophthalmoscopic examination during the occurrence of the symptoms. On this latter point I am able to confirm the observations made by Foerster, Haskett Derby, and others. The occasional occurrence at the same time of other cerebral symptoms is another circumstance which is suggestive of a central origin. An interesting experiment, too, which points to the same conclusion was made by Kums. He found that phosphenes produced by pressure could not only be perceived during the attack, and whilst the subjective sensation of light was at its height, but that these were referred to a different plane from that to which the scintillating scotoma was projected. It seems probable, though, that when the hemianopic or bilateral character is not marked the disturbance may in some cases really proceed from the retina. Thus I have occasionally met with excentric negative scotomata in one eye only which appear to have been the result of attacks of this nature. It would be interesting to try the result of Kums' experiment during the attack in such cases. Another kind of

central subjective coloured vision, which is probably much rarer than that connected with hemicrania, takes the form of an aura preceding an epileptic fit. This I have not seen, but it is described by Hughlings Jackson, and seems most commonly to be an erythropsia, everything appearing to the patient intensely red.

Myodesopsia, etc.—The appearance of shadows in front of the eye may or may not be pathological, according to their nature and the conditions under which they are seen. There are many irregularities in the normal eye which interfere to a slight extent with the passage of the light rays to the retina, and therefore cast shadows on it. These shadows are, however, mostly too faint to be perceived owing to the small size of the bodies which throw them compared to the extent of the surface of light from which rays pass through the pupil, the shadow thrown by one point of light being illuminated by the rays proceeding from others. Only such bodies as lie very close to the retina are rendered at all visible by their shadows, and even then are, as a rule, so faint as to escape observation. Many people notice small faint shadows which they project to different distances in front of their eyes when looking at a uniformly illuminated surface, such as a white cloud or sheet of white paper. These have different shapes: they are annular or strung together in beaded chains, or have more the appearance of irregular shreds of tissue. They are not, as a rule, fixed, so that while following the movements of the eye they are generally observed to change their position as soon as the eye is brought to rest. On looking upwards, for instance, they appear first to be thrown up along with the eye. On this account, these small faint shadows are called muscæ volitantes. The objective cause for them is the existence in the vitreous chamber of small portions of the embryonic tissue of the vitreous body. The fact that these shreds of tissue throw shadows at all, under ordinary circumstances, shows that they must lie at the posterior part of the vitreous. Owing, too, to the free movement of these muscæ, the vitreous must be more or less fluid in the portion occupied by them. As, however, the same muscæ can always be seen, over and over again, pretty much at the will of the individual, and

differ very little in their faintness, the fluid portion on which they float must be very narrow. When the rays which enter the eye proceed from a luminous source of very small extent, such as is the case with those which pass through a pin hole in a card held close in front of the eye, the muscæ appear much darker and more numerous, and other more anteriorly placed irregularities become, at the same time, visible. It is not easy to draw the line between what may be looked upon as pathological in respect to the appearance of muscæ volitantes and what is merely physiological. With a small pupil and continued fixation, such as is necessary for writing or drawing on strongly illuminated sheets of white paper, etc., the muscæ seldom fail to be observed, though they are more readily seen where there is myopia and the surface looked at lies beyond the far point. When attention is once drawn to them they frequently cause considerable annoyance. Yet, under such conditions their appearance cannot be considered other than physiological. On the other hand, when the conditions are not specially favourable, and they yet cause more or less constant annoyance, they are an indication of the existence of a hyperæsthetic state of the retina, and as such often of some general disturbance, most frequently in connexion with the liver or other digestive organs. When numerous and changeable, they point to an abnormal degree of fluidity of the posterior part of the vitreous; and such cases are often associated with the higher degrees of myopia. As long, however, as the bodies casting the shadows are so small as not to be recognisable on ophthalmoscopic examination, they may be generally diagnosed as mere muscæ. The larger floating opacities in cases of disease of the vitreous or hæmorrhage into the vitreous are visible ophthalmoscopically, so that the complaint of anything appearing to float in front of the eye should always lead one to make a proper objective as well as subjective examination.

CHAPTER VI.

DEFECTS OF THE LIGHT AND COLOUR SENSES—AMBLYOPIA AND AMAUROSIS.

WHILST the pathology of the senses of form and colour has long been studied, it is only recently that the abnormalitics of another important function of the retina, viz., the sensc of light, apart altogether from regional quantitative and qualitative differences—of the light sense pure and simple—have received attention. Any attempt at obtaining a method of subjective examination, whatever its nature, which shall admit of mathematically accurate comparativc measurements must necessarily fail; it is sufficient that the limits of error should not transgress the bounds of practical utility. It has probably been greatly on account of this fact not having been kept in view, as well as perhaps owing to the undoubted difficulty of clearly distinguishing bctween pure sensations of light and mixed scnsations of light and form, as they all but invariably present themselves to our consciousness, that the light sense had until recently received so little development from a pathological point of view, although many points in the physiology of light perception have long been known. The only systematic investigations made before 1883 were those of Foerster and his pupils. Foerster determined in different diseases of the eye, and by means of his photometer, the degree of absolute illumination required to render a particular object only just visible. This degree of illumination, compared with the normal standard under similar conditions, furnishcd him with a fraction which he took to represent the acuity of the light sense in any particular case. Some important results were got from these investigations, principally with reference to the symptoms of syphilitic choroiditis, for whieh disease the enormous diminution of the light sense tested

by Foerster's or any other similar method is all but characteristic. An important advance was made by Bjerrum in 1883. His investigations led him to the discovery of a point of considerable diagnostic importance, and consequently directed more attention to the whole subject. The two points on which it is important to gain information are,—(1), The amount of illumination which is just sufficient to give rise to a sensation of light; and (2), The smallest difference between two intensities of illumination which is capable of being distinguished. The values of these two factors depend on the visual angle, so that the smaller the angle the greater is the fraction expressing them. Further, the value of the latter depends (within certain limits) on the absolute intensity of the light under which the test is made. Bjerrum found that deviations from the normal conditions of the light sense only took place in two directions,—(1), That in which the minimum amount of light perceptible is represented by a much greater absolute quantity than normal, whilst a normal power of distinguishing between impressions of different intensity remains; and (2), That in which the fraction representing the delicacy of perceiving differences of intensity is greater than normal, whilst the minimum of light appreciation does not materially differ from the normal standard. Diseases primarily involving the choroid and retina show more or less tendency to a deviation in the first respect; those primarily involving the nervous elements in the retina or optic nerve to a deviation in the latter respect. After experimenting with various methods for the determination of the two distinct factors composing the light sense, Bjerrum decided that the most practical instruments were what are known respectively as Foerster's photometer and Masson's disc. Foerster's photometer consists of a rectangular box one foot long, and rather less than half a foot in breadth and height, blackened inside, and fixed to an adjustable stand. To one side of one of the ends of this box two apertures prolonged outwards into short projecting tubes are placed for the two eyes to look through. At the side of these apertures is the arrangement for illuminating the interior of the box. This consists of a standard candle, kept always at the same height by means of a spring, and placed at a

short distance from a hole or window in the box which is covered with white paper. The size of this paper window, and consequently the amount of light which enters the box, is regulated by an adjustable rectangular diaphragm, the centre of which remains always in the same position. By knowing the area of this diaphragm it is easy to calculate the corresponding degrees of illumination. At the other end of the box is placed the object to be recognised, most conveniently a couple of squares of white paper about one inch apart, and also about one inch in size.

A difficulty in connexion with all methods for determining the minimum perceptible quantity of light arises from the enormous degree to which that quantity varies according to what is known as the state of adaptation of the retina. The sensitiveness to light is indeed many hundred times less when the eye has been exposed for some time to strong daylight than it is when it has been for some time entirely kept in the dark. Practically, then, the unit must be taken from a comparison with one's own light perception under the same conditions and with proper care that the patient examined understands the nature of the test which is being made.

Masson's disc consists of a disc of white cardboard, on which is painted a number of black sectors of different sizes. These black sectors produce, on rapid rotation of the disc, a number of grey rings, the intensity of which varies. Thus the difference in intensity between the light reflected from the white surface alone, and the grey ring produced by the combination of the impressions from it and a black sector of 4°, is practically $\frac{1}{90}$; with a sector of 6° it is $\frac{1}{60}$, and so on. Although the normal power of distinguishing between degrees of intensity varies with the absolute illumination, the amount of such variation is slight, and does not cause any difficulty in examination in which one has always, of course, the contrast furnished by what one sees one's self. The rings should have a considerable breadth (about $\frac{1}{2}''$), so that their visibility at 1 foot is independent of the acuity of vision in any case where the test is at all applicable. Independent researches made by Samelsohn have led in the main to a confirmation of

Bjerrum's results, though there are some minor points on which they disagree, which appear to be due to the less accurate methods of examination used by Samelsohn.

The pathology of the light sense seems likely to afford a clue as to the nature of some little understood affections. For instance, the result of an examination into the two factors of that sense in cases of glaucoma is that either a marked diminution takes place in both, or, at all events, that in that disease the minimum perceptible quantity of light is very much greater than normal. This certainly points to the choroid as the site of the affection. Again, the condition of the light sense in the amblyopic area in toxic amblyopia is closely similar to what is found in cases of optic neuritis and atrophy. This is suggestive when taken along with the recent views on the etiology of this affection.[1] In a paper communicated by Samelsohn to the ophthalmic section of the last International Medical Congress, he sums up the conditions in which, so far as our present knowledge goes, an examination of the light sense is likely to be of diagnostic or prognostic significance. They are—(1), In certain forms of dense opacities of the vitreous, when there might be a doubt as to the existence or not of detachment of the retina. In such cases, if the power of distinguishing between different light intensities is not diminished, there is not likely to be any detachment; (2), In cases where the differential diagnosis between glaucoma simplex and optic atrophy presents great difficulties, a relatively great diminution in the power of perceiving feeble illuminations, combined with a relatively small interference with that of distinguishing between different intensities of light, is suggestive of glaucoma, the opposite condition being, as we have seen, suggestive of atrophy; and (3), In the case of immature cataract, where there is any suspicion of the visual acuity not corresponding to the degree of opacity of the lens, the examination of the second factor of the light sense is of importance in enabling one to exclude the likelihood of a complication with optic atrophy.

[1] *Vide* Chap. VII.

Colour Sense. — Congenital defects of colour vision occur in quite 3% of the male population of civilized countries. Amongst females the percentage is enormously much lower, or only about $\frac{1}{20}$ of that for males.

This comparative frequency of defects of colour vision has directed attention to the possible dangers which might result from the employment in our railways and mercantile fleet of individuals unable to distinguish with certainty between the colours universally used as signals, viz., red and green.

Accidents directly traceable to mistakes arising from colour-blindness must be of extremely rare occurrence, and the possible dangers have certainly been considerably exaggerated. Still there can be no doubt that a systematic examination of the colour vision of all persons entering these services is desirable.

When the possible dangers in connexion with colour-blindness were recognised, it naturally became of importance to discover a means whereby any trace of this anomaly could be speedily detected. Holmgren[1] has undoubtedly the merit of being the first to devise and employ a method which has shown itself to be thoroughly practical and expeditious.

It is a well-known fact that a high degree of achromatopsia may co-exist with a tolerably perfect power of naming colours, from which it is evident that any system based on the statements made by individuals as to the names of colours presented to them must be rejected as impracticable. Holmgren has therefore adopted the method of *comparison* between colours which to the normal eye are different, a method which, in a less perfect manner, inasmuch as the time required for examination is much longer, was used by Maxwell and Seebeck. Holmgren's method is, in fact, a modification of Seebeck's: the individual examined is asked to pick out from amongst a large number of differently coloured wools those which appear most like one particular shade placed before him. The reasons for choosing wool instead of coloured glass, paper pigments, etc., are, that all colours and shades of wool are easily

[1] *Om Färgblindheten i dess förhaallande till Jernvägstrafiken och Sjöväsendet.* Upsala, 1877.

obtained; that it can be used without any preparation as obtained; that, having the same colour on all sides, it is easily recognised amongst a lot of other coloured objects; that as its surface is rough, no difficulty is caused by reflection; and finally, that it is easily packed and carried about.

From the way in which this test is executed by different individuals, it can at once be seen whether they are colour-blind or not: those with normal vision, provided they are possessed of a certain amount of intelligence, are not long in selecting the few shades which most nearly resemble the pattern given them to match; and a colour-blind individual soon commits a sufficient number of mistakes to amply reveal his defect. In order, however, at once to obtain some idea as to the nature of his colour-blindness, it is advisable to choose certain colours as patterns Holmgren begins with light green, and, when mistakes are made with this, proceeds with some shade of rose or purple, which, owing to its position in the scale of colour perceptions,—viz., between red and blue,—is very well suited for this purpose, as it can at once be seen from the colours with which it is confounded, or, in other words, with which it is pseudo-isochromatic, in which direction the defect lies.

Several subsequent investigators have rejected as superfluous the preliminary examination with light green, and begun at once with the rose colour. This is a mistake, as, although rose probably suffices as a test for all forms of colour-blindness when complete, yet slight anomalies of colour vision are most easily detected by using light green. Holmgren gives, as the result of examination by his method, the following classification of all cases of defects of colour vision:—

I. *Total Colour-blindness.*—In these cases colour hues are not distinguished from each other as such, but only according to their relative brightness (very much in the same way as the normal eye would distinguish coloured objects illumined by a sodium flame alone).

II. *Partial Colour-blindness.*—This may be (*a*) complete or (*b*) incomplete.

Complete partial colour-blindness he divides into (α) red-blindness, (β) green-blindness, and (γ) violet-blindness.

Although most writers are agreed as to the great superiority of the above described method for the rapid determination of colour-blindness, there is a want of unanimity with regard to the classification of cases of partial colour-blindness; some, amongst whom are Hering, Cohn, Stilling, etc., contending that there is no difference between red and green blindness, and between blue and yellow blindness, whilst others—Donders, Raehlmann, Magnus, etc.,—follow Holmgren's classification, which is based on the Young-Helmholtz theory of colour perception.

Now, what is the actual condition of the sense of colour in those who are colour-blind? In the first place, there can be no doubt that an individual who is blind for one particular colour hue is at the same time blind for its complement. That this is the case is shown by the following facts:—It is possible, as is well known, by the rapid rotation of a disc, to obtain from three or more suitably coloured sectors an impression which is identical with that of a mixture of white and black produced in the same way: the colours, taken in certain proportions, can be got to neutralize each other, so that the resulting impression is colourless. The slightest change, however, made in the proportion of each colour, or the removal of any portion of one of the colours entering into the combination, can at once be detected, and some colour sensation is the result. If, on the other hand, the disc should contain two sectors of exactly complementary colours, their simultaneous removal does not destroy the colourless effect; the remaining colours continue to neutralize each other, so that the impressions they give rise to, following each other in rapid succession, resolve themselves into grey. Now, it is found that the same mixture which to a normal individual appears similar to a mixture of black and white, appears also to the colour-blind identical with the same grey thus produced; whence it follows, as they are known to be blind for one colour, that they must either be insensitive to two complementary hues in both discs, or to only one in each. If they only fail to perceive one, both discs must appear to them

coloured; but this is unlikely, because then all objects which appear to the normal eye colourless must appear to them coloured, which there is no reason to suppose is the case. But there is another reason for believing that this cannot be the case, viz., the analogy which exists between physiological colour-blindness at the periphery of the retina and the normal colour-sense on the one hand, and pathological colour-blindness on the other.

Adamük and Woinow have found that the colour mixtures which appear grey at certain parts of the periphery of the retina vary according to the intensity of the illumination; and, according to Donders and Landolt, the peripheral colour impressions do not differ from the central if the intensity of illumination be increased.

Although the colour-blindness existing for the peripheral parts of the field of vision is only partial, still the fact remains that there a very similar colour confusion exists as in the colour-blind. Thus a red or green object will appear, when viewed peripherally, yellow, grey, or blue, according to the hue taken; yet a white or grey object does not become coloured by being moved from the centre to the periphere of the field of vision, which, as a white surface reflects all rays equally, or at any rate nothing but rays which neutralize each other, would necessarily be the case if certain parts of our retina were insensitive only to particular and non-complementary homogeneous rays.

Again, if a certain colour appear colourless to a colour-blind individual, its after-image appears also colourless, and one which fails to produce the normal impression also gives rise to an after-image which is exactly complementary to the impression received, and not to that which would be seen by any one whose colour-sense was not defective. There is no reason, however, why rays which, although colourless, are yet visible, should fail to produce in us the usual successive or simultaneous contrast, unless we are at the same time insensitive to those which give rise to the complementary impression; indeed, if we have not the power of evoking the normal impression subjectively, we cannot expect to do so objectively.

Another point of importance in connexion with the vision of the colour-blind is, that although they fail to distinguish between many different colours, yet they are only actually blind for two particular hues which are complementary, and the slightest change in which is capable of giving rise to a colour impression. Thus most see a continuous spectrum: yet the colours seen are but shades of two hues which are separated by a narrow band of grey. This band, too, diminishes in breadth according as the intensity of the illumination increases. These *neutral* points appear, however, to differ in different cases of colour-blindness, which, nevertheless, are generally included under the same class. If, therefore, we could imagine all our possible colour hue perceptions so disposed round the periphery of a circle that those which are complementary were exactly opposite each other, we should find that the directions of the diameters representing the exact hues for which different individuals were blind differ. Such a circle would necessarily include not only all the different homogeneous light rays which we are capable of distinguishing as different hues,—in other words, all the colours of the spectrum,—but also such complements to these colours as are not contained in the spectrum, which for some reason or other we are not able to see as homogeneous light, although we can do so subjectively, or by mixing other spectral colours.

The reason why only one neutral line is usually to be found in the spectrum of the colour-blind is, that in the immense majority of cases the diameter representing the hues for which they are insensitive passes from some part of the green to some part of the space which would be occupied by the purplish hues invisible as homogeneous colours.

The position of this colour-blind diameter *has an influence on the perception of all the colours of the spectrum*, and constitutes a difference—at one time small, at another time considerable—between two cases of colour-blindness, so that if in one case the diameter lie between bright green and purple, the colour confusion will differ from that presented by a case where it lies between a more bluish green and a more reddish purple or rose colour. This,

we believe, is the true explanation of the difference between green and red blindness of some authors.

Many investigators describe the spectrum of the colour-blind as continuous although only containing two colours, no portion appearing grey. This is due to the way in which they have conducted their examinations. If a very luminous spectrum be used, the images of the slit formed by the rays lying on each side of the ones giving rise to the exact neutral line so overlap each other as to cover the image formed by these colourless rays. A less luminous spectrum, by diminishing the colour sensations produced on each side of the neutral line, which, in all cases, are described as feeble, gives rise to the impression of a grey line separating the two colours composing the spectrum. Cases, again, do occur in which no neutral line is observed. These are cases of incomplete colour-blindness which are not infrequent, and of very different degrees.

Again, if we take any red, orange, yellow, or green sector, and combine the impression received from it with that from a blue pigment by rotation on a disc, we find that the proportions of any of these with the blue, which is necessary to produce a neutral colour sensation, varies in different cases of colour-blindness, that is to say, different cases are blind for different non-saturated hues—hues which owing to their mixture with white light are impure, and owing to the absorption of light by the pigments are wanting in brightness. In all probability, therefore, there are a great number of forms of complete colour-blindness corresponding in general to blindness for certain rose and green hues; and although the classification into red-green-blindness and blue-yellow-blindness is preferable to that based on the theory of three fundamental sensations, still there can be little doubt that a more accurate one might be taken from the hues representing neutral sensations.

The explanation of why, in the case of the partially colour-blind, the absence of the perception of two complementary hues should leave the individual only a dichromatic spectrum has been attempted in different ways, and has led to the various hypotheses of colour perception, none of which can be said to be more than

extremely speculative. Cases of congenital colour-blindness of one eye alone have been met with, and these have afforded opportunities of studying how the different colours appear to the colour-blind eye when compared with the normal impressions of the other. It is very rare indeed for colour-blindness to be acquired unless there is, at the same time, a marked defect of visual acuity and a considerable diminution of the field of vision.[1]

Amblyopia and Amaurosis.

When the visual acuity, after correction if necessary of any existing error in refraction, does not come up to the normal standard, there is said to be amblyopia. The term amaurosis should be kept for complete, or almost complete, blindness. Normal vision suffices for the discrimination of two objects which are separated by a space, making, with the nodal point of the eye, an angle of at least 1′, provided the absolute illumination, as well as the difference in illumination of objects and interspace, are sufficiently great. Amblyopia may be the result of defects in any part of the visual apparatus, and may be congenital or acquired. It is seldom that the mere subjective examination of the visual acuity is sufficient to lead one to the diagnosis of the cause of the amblyopia. Most frequently other subjective tests for the determination of the condition of peripheral vision, of the colour and light senses, etc., have also to be made; or where the cause is superficial, it is often more or less apparent on inspection. Faint nebulæ of the cornea are a common cause of slight degrees of amblyopia, and these are often only first recognised on oblique illumination. Irregular astigmatism, too, and conical cornea, are amongst the defects in the anterior part of the eye which cause more or less amblyopia, and which when slight are not always easily detected. Defective vision, when congenital or originating early in life, often escapes observation, unless the degree is considerable, until the difficulties experienced at school attract attention. Lamellar cataract, for instance, is rarely discovered until the period of school life, and even then frequently fails to be

[1] *Vide* Chap. VII.

recognised as the cause of the amblyopia, which is attributed entirely to the real and apparent myopia which often coexists. The higher degrees of congenital amblyopia are mostly associated with nystagmus. It is by no means an uncommon thing to find very considerable amblyopia and nystagmus without any marked objective cause, either in the dioptric or percipient parts of the eye. In such cases there is usually a history either of early severe inflammation of the cornea, followed by dense opacitis, which have slowly cleared away, leaving little or no trace; or of some cerebral affection, which has interfered more or less completely with the visual centres, and which has been followed by only partial recovery. In both cases the conditions have been unfavourable to the acquirement of that superiority of the vision of the centres of the retinæ, or maculæ luteæ, over the other parts, which characterizes more normal vision and admits of the usual steadiness of fixation. Congenital amblyopia, ranging from the slightest defects of vision to that where the acuity is but one-tenth or less of the normal and unaccompanied by nystagmus, is very frequently met with without there being any objective sign or any history to account for it. Frequently, too, the degree of amblyopia where there *is* some defect is much greater than can be accounted for by the defect alone.

When amblyopia is present only in one eye, it is still more likely to pass unobserved, unless, as is frequently the case, it gives rise, owing to the existence of other predisposing causes, to strabismus. Often a unilateral congenital amblyopia is only accidentally discovered late in life, when some trifling accident to the eye, it may be, has drawn the individual's attention to it. Amblyopia is sometimes simulated. In this country it is rare, comparatively speaking, to meet with this, and the deception is seldom so well carried out that there is much difficulty in detecting it. In other countries, where there is a compulsory military service, simulated partial or complete blindness is very frequently met with. There may be a simulation of either bilateral or unilateral defect of sight. The latter is by far the most common, and fortunately, too, it is the most easily detected. More fre-

quently it is an amblyopia, and not a complete blindness of the one eye which is feigned, so that the diagnosis may not be at all easy. When bilateral amblyopia is pretended, a little ordinary care on the part of the individual is all that is required to make it almost impossible for one to detect it with certainty. Yet it is strange how often some inconsisteney between the acuity of vision found on examination for distanee and for near, or in connexion with the glasses which are admitted to effeet some improvement in vision, affords a elue to the eharaeter of the amblyopia. I have, for instance, repeatedly, and more especially in young girls, obtained very considerable improvement of vision by the use of a convex and concave glass whieh exactly neutralized eaeh other. It requires, on the other hand, a very great amount of eare and considerable knowledge of the subjeet to simulate well monocular amblyopia. The eertainty of the diagnosis depends pretty mueh, too, on the degree of the pretended blindness. A great number of tests have been devised for the deteetion of this kind of deeeption, all of which it is probably possible to elude. An easily applied test is that whieh ean be made with Snellen's eoloured letters. These are transparent red and green letters of different sizes. The patient is first made to read them out without anything before his eyes. A pair of reversible speetaeles, one eye of which is of green glass and the other of red, is then put rapidly upon his nose, eare being taken that the eyes are all the time kept open. As the green glass entirely exeludes all the rays from the red letters, and the red all the rays from the green, some of the letters are visible only to the one eye, and others only to the other. Any mistake made in the reading of the letters is in this way easily deteeted. The slightest blinking of one eye is sufficient to show one with which eye each partieular letter is seen, so that with care it is possible for a elever deceiver to avoid falling into this trap, although the manner in which the individual behaves may be generally sufficient to arouse or confirm one's suspieions. Another simple test in cases where complete, or almost eomplete, blindness of one eye is simulated consists in holding a prism with its base directed upwards or downwards in front of the good eye.

If this produces diplopia, there is evidently vision in both eyes; but the object of this test, again, is of course easily defeated by any one denying that he sees double. An important modification of the test is made by Alfred Græfe. By causing the individual to be tested to shut the blind eye, and holding the prism in front of the good one in such a position that some of the rays pass into the pupil through the prism, and some directly, monocular diplopia is produced. The patient, thus thrown off his guard, often admits that he still sees double when the blind eye is uncovered and the position of the prism altered to the slight extent required to intercept all the rays passing into the pupil of the seeing eye, under which circumstances the diplopia is of course binocular, and its existence is a proof of vision in the eye said to be blind. Von Græfe's well-known test of placing a prism with its base directed inwards or outwards in front of the seeing eye, and observing whether a lateral movement of the eye is thereby induced, is perhaps the best test for simulated amaurosis of one eye, because where binocular vision exists such a movement almost always takes place involuntarily, owing to the unconscious desire for the fusion of the two images.

A good number of cases of amblyopia and amaurosis occur more or less suddenly, and unaccompanied, at the time at least, by any ophthalmoscopic changes. In such cases the diagnosis has to be made from a consideration of the history. When the blindness comes on in the course of a severe illness or during the period of convalescence, for instance in scarlet fever, puerperal albuminuria, etc., the cause is most often uræmic and the prognosis favourable, more especially if the pupils remain active. Amblyopia or amaurosis occurring after severe hæmorrhage is very unlikely to be recovered from, and is generally followed after some time by optic atrophy. The prognosis is even worse in cases of unilateral amaurosis after a severe fall on the head. These cases are of pretty frequent occurrence, and are probably almost invariably due to laceration of the optic nerve or hæmorrhage into the sheath of the nerve, owing to fracture of the roof of the orbit, the line of which fracture usually passes through the optic foramen.

The atrophy to which this lesion gives rise is not visible ophthalmoscopically until after some time. The blindness which one meets with in young children after severe cerebral symptoms is often unaccompanied by ophthalmoscopic changes, and is due, no doubt, to interference with the functions of the visual centres. It is often partially recovered from; but even in cases where the pupil remains active I have seen permanent blindness remain. The rare cases of blindness from lightning do not seem to have received a proper explanation as yet. Only one case of this nature has come under my own observation, and that after complete recovery. In this case, which was kindly brought to my notice by Dr Burn-Murdoch, there was complete amaurosis, lasting for six weeks, with ptosis. On the whole, judging from the recorded cases, the prognosis seems favourable. Perhaps the most interesting cases of amblyopia without ophthalmoscopic changes are the reflex and hysterical forms. Reflex irritations may proceed from the teeth, stomach, or intestines, or from the uterus and ovaries, and the amblyopia to which they give rise, after persisting for months, often disappears quickly on removal of the source of irritation. Very little is known of the more exact pathology of these cases. They are vaso-motor disturbances, no doubt, but whether of the retina or of the centres of vision is not very clear. The former appears, however, the more likely, as they frequently exhibit the symptoms of anæsthesia or hyperæsthesia of the retina, the fields of vision, of both eyes as a rule, being concentrically restricted sometimes to a very extreme extent, while the central vision is not so very much impaired. In some of the more distinctly hysterical cases, too, there appears to be a considerable improvement in peripheral vision in subdued light.[1] Cases of complete hysterical amaurosis are the most extraordinary, both in their symptoms and in the suddenness with which, under certain strong emotional excitements, they may be recovered from. There is in such cases evidently a withdrawal of the attention from the impressions received by the visual centres, whilst at the same time a kind of more or less perfect unconscious perception of them remains.

[1] *Vide* Day Blindness, p. 49.

CHAPTER VII.

LIMITATIONS OF THE FIELD OF VISION.

THE examination of the field of vision is capable of affording indications of great importance from a diagnostic as well as prognostic point of view. The periphery of the retina, though not to the same extent as its centre, may receive three distinct impressions—those of light, colour, and form. The first is a quantitative, the second a qualitative impression, and the third may be looked upon as the relative appreciation of differences in either or both of the other two, affecting simultaneously, or at sufficiently close intervals of time, anatomically distinct portions of the retina.

The abnormalities of the light sense of the peripheral portions of the retina have not as yet received very much attention, partly because it is difficult to conceive of a practical means of testing it which does not at the same time involve the sense of form, and partly because the physiology of the subject is still imperfectly known.

Abnormalities of the colour sense have been more studied; they will be considered further on.

Peripheral vision may be defective in continuity or in acuity. The continuity is subject to both regular and irregular interruptions. The regular manifest themselves by some limitation of the normal extent in a particular, or, it may be, in all directions. The irregular breeches are more or less blind portions, surrounded by normal, or relatively normal, portions of the field of vision. To them the name of *scotomata* has been given. To make an accurate examination of the field of vision, it is necessary to make use of some sort of perimeter. A rough examination, which is sufficient for the determination of any existing limitation in the peripheral boundaries of the field, can be made in the following manner:—The patient is placed with his back to the light and

facing the observer. He is directed to cover one eye with his hand, and to fix steadily with the other the eye of the observer which is directly opposite his own; therefore if the left be the one to be examined, the patient fixes with his left eye the right eye of the observer, whilst the observer, at the same time, fixes with his right eye the patient's left. In this position there is obviously a plane at right angles to the common line of fixation and cutting that line at its mid-point; any point on which provided rays from it enter the two eyes will have an image at correspondingly situated points on the two retinæ. If, therefore, the observer on moving his hand in this plane from or towards the middle of the line of fixation finds that it disappears from or comes into his own and the patient's view simultaneously in any direction, it follows that their fields of vision are co-extensive in that direction. A test in this manner can be rapidly made in all directions, and a want of coincidence of the two fields in any or all directions be easily discovered. This method of testing is only, however, a qualitative method, in so far as by it the existence and not the extent of a limitation is determined. It is besides inapplicable, as a rule, to cases where there are interruptions in the field. Of perimeters the most convenient are what are called self-registering perimeters, with which instruments a record or chart is taken at the time of the examination.

In the charts ordinarily used, the centre of the figure (*vide e.g.*, Fig. 4) corresponds to the point of fixation, whilst the concentric circles represent the positions occupied by objects whose retinal images are equidistant in all directions from the macula. Fig. 2 represents Blix's self-registering perimeter, which is both practical and inexpensive. The instrument is fixed to the back of a chair, on which the individual sits with his head resting against the support A. The eye comes then to occupy a position on the vertical line below B and on a level with D. D is a small mirror or white object which enables the individual examined to maintain a steady line of fixation. A small white or coloured disc on a piece of black cardboard C is used as the object for which the field of vision is taken. The position shown in the figure, in

which C and D are in a line with the eye, is the starting position. The white disc on C is then opposite the macula (though hidden

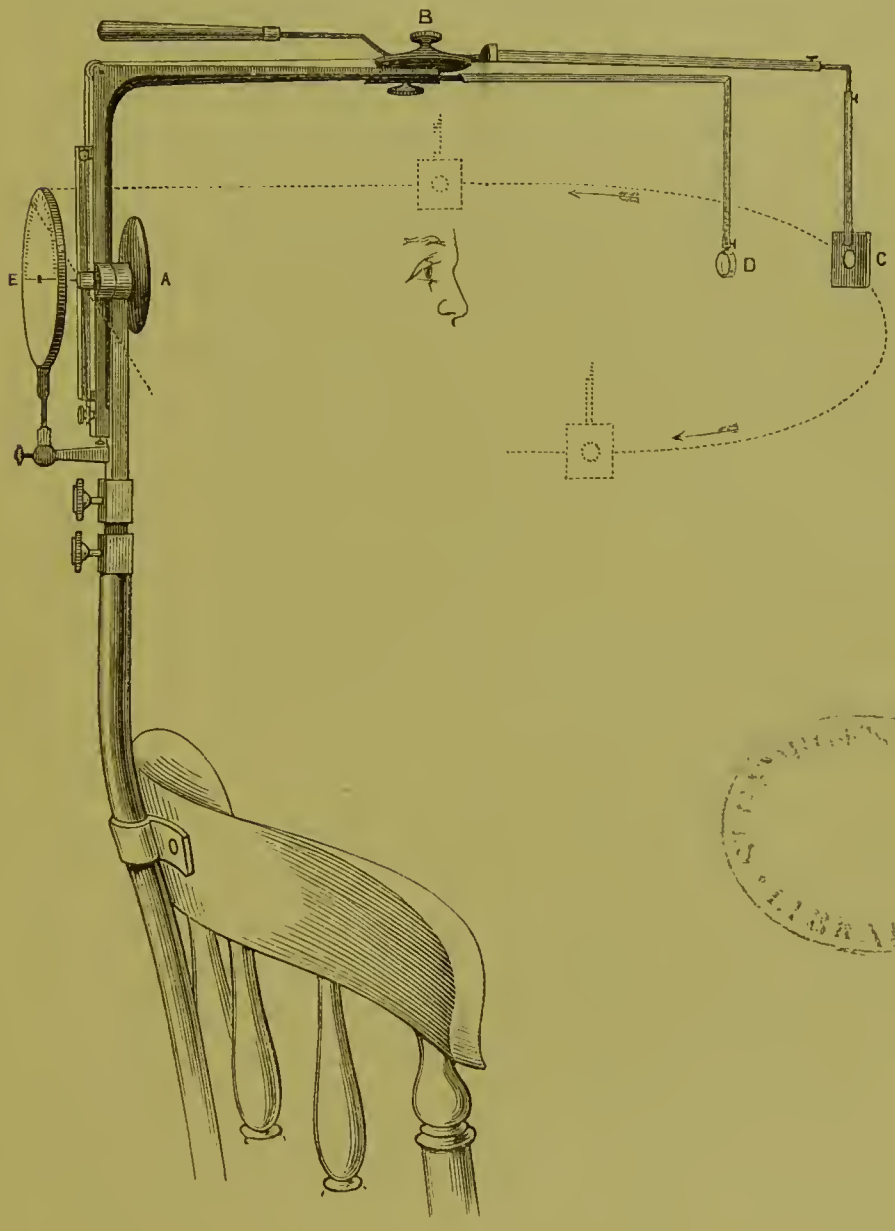


Fig. 2.

by D), and therefore occupies the centre of the field of vision. C is moved in a circle round the eye as centre by means of the handle In the drawing this movement is shown as taking place in a horizontal plane. The plane of the circular path of C can, how-

ever, be varied at will, so that C may be made to occupy any possible position on the surface of a hemisphere round the eye by turning the upright arm round an axis at the joint behind A. A register of the position of C for different meridians of the field is got on a chart fixed on E by tapping E against a little movable stilette (see Fig.) The movement of this indicator takes place along the steel bar attached to the upright (and movable with it round the axis below A) by means of a piece of harp string which passes round a groove in the flat wheel below B, and to which it is firmly fixed at one point, then over two pulleys at the angle between the upright and horizontal portions of the instrument.

If there be any doubt as to the restriction of the field in any particular case, the examination is best made by artificial light, the intensity of which is a little greater than that which begins to tell on the normal field. This is a sort of compromise between the examination of the light and form senses which is of considerable practical importance. The white object used in the examination should not be larger than 10 mm. square, and the observer should stand in such a position as to make sure of the patient constantly fixing the centre of the perimeter.

The normal extent of the field of vision is subject to variation at the upper, and slightly also at the inner parts, owing to individual peculiarities in the size and shape of the eyebrows and nose. The physiological limits may be taken to be as follows:—Upwards, 45°; upwards and outwards, 50°–55°; outwards, 90° (often slightly more); outwards and downwards, 80°–85°; downwards, 70°; downwards and inwards, 60° (variable on account of the nose); inwards, 55°–60°; inwards and upwards, 55°. The extent of the field upwards and upwards and outwards is found to be 5°–15° greater when the point of fixation is situated 20° or 30° from the centre of the perimeter in the opposite direction. This has to be borne in mind when there is a doubt as to whether or not there is limitation in this region. Thus, should the exploration with the centre of the perimeter as point of fixation only give an angular dimension of 40° for the peripheral extent of the field upwards, we should not always be right in assuming a contraction in this

direction unless *no increased measurement* resulted from testing with a lower point of fixation.

The determination of the limits of the field for different colours is a matter of much greater difficulty and uncertainty, as the results are influenced by the hue and shade of the colour used, by the size of the coloured objects, and by the quality and intensity of the light under which the examination is conducted. Practically, in examining any case, I find it useful to determine the extent of my own visual field in one or two directions under the same conditions, and then allow something for individual peculiarity besides. Another precaution that will be found useful in practice is to have the test object (which it is well to make 20 mm. square) differently coloured on either side. In this way we are provided with a check on the accuracy of the patient's statements. The most convenient colour to use is some hue of red, as it is for the reds and greens that pathological defects in the colour sense first manifest themselves. If the test colour chosen be examined carefully at the inner side of the field of vision, it will generally be found to change colour. Before becoming absolutely colourless, it will become yellowish, brownish, or bluish, according to the hue and shade selected, and it is this tint which may with advantage be selected for colouring the opposite side of the test object. When the red-green perception is entirely abolished, the limits may be taken for blue or yellow, the vision for which is longer in disappearing, but the only necessity for this is to exclude the possibility of the more usual form of congenital colour-blindness, which we should have reason to suspect if the peripheral boundaries for yellow and blue were not restricted. But the determination of the limits of the field for quantitative and qualitative stimuli does not exhaust the examination of indirect vision: it is important to ascertain whether or not there is any break in the continuity of the field. Such interruptions, or scotomata, are usually divided into *positive* and *negative*, according as they give rise or not to a consciousness of the interruption. In the one case there is more or less perception of darkness, in the other an entire absence of any visual impression. The scotoma

existing over the projection of the area of the optic nerve where it enters the eye is a familiar example of the negative variety, though it differs in some respects from those which arise pathologically (viz., in the manner in which the impression elicited by excitation of the retinal elements round about are mutually related).

Where a lesion primarily involves the nerve fibres of the retina the resulting scotoma is unperceived, or *negative;* where, on the other hand, lesions involving the retinal pigment and choroid are the cause of the defective area, there is more or less consciousness of its presence. This Foerster explains by the assumption of an uncqual stimulation of the light-perceiving elements in the defective and surrounding areas, the subjective sensation associated with any lesion giving rise to torpor of the retina being that of relative darkness. Positive scotomata are generally most marked in subdued light, and are sometimes so distinct that the patients are able to draw them as they are projected on a white piece of paper held in front of them.

The conditions in which some alteration or restriction of the field of vision occurs are: optic neuritis and atrophy, the funetional forms of amblyopia (under which group are included the various forms of amblyopia *sine causâ*, such as toxic amblyopia, amblyopia fugax, the various conditions connected with hysteria, etc.),[1] retinitis, more especially retinitis pigmentosa, embolism of the central artery, and thrombosis of the central vein of the retina, opaque nerve fibres, glaucoma, detached retina, intraocular tumours and foreign bodies, choroiditis, and coloboma of the choroid.

Field in detached Retina.—The portion of retina detached can be seen with the ophthalmoscope, but it is impossible in this way, even with careful drawing, always to form an idea of the extent of the lesion sufficiently exact to be of use in comparing the conditions met with on the first and subsequent examination of any case. The most trustworthy means of making such a comparison is by plotting out the field. It must be borne in mind, however, that the freshly detached retina retains its functions to a slight

[1] *Vide* Chap. VI.

extent, so that it is always advisable to make the examination in subdued light, of course under as nearly as possible identical conditions each time. Were a conscientious examination always made in this manner, there can be little doubt that cases of recovery from detached retina would very seldom be recorded. The boundaries for peripheral colour vision are narrower than the line separating the undetached from the detached portion of the retina, so that an examination with pigments might be made instead of the ordinary one, which, however, is sufficiently accurate if the precaution of using a very subdued light be attended to. In cases of detached retina there is often a remarkable confusion between blue and green. This was first observed by Leber, and ascribed by him to absorption of the blue rays by the yellow sub-retinal fluid. Certain points in connexion with the form and method of extension of the detachment, as well as of the symptoms to which it gives rise, are sufficiently characteristic to engage attention. Thus the defect in the field of vision has most frequently a more or less indefinite, ill-defined, and irregular boundary. If the field be restricted below, an extension upwards is not unlikely to follow. This may pass to either or both sides of the point of fixation, though generally it does so to the inner alone, or it may involve the centre itself in its course. Such an extension is not so likely to be the case where the first restriction, as often happens, makes its appearance above. In almost all cases of simple detachment of the retina the central visual acuity is more or less diminished, indicating some participation by the region of the macula lutea in the disease which has led to the detachment elsewhere ; a condition, in fact, of œdema which sometimes improves, and is apt to lead to the belief that the detachment itself has been recovered from. Often the subjects of detached retina are conscious of a certain degree of distortion of the images which they receive,[1] and it is not uncommon to hear them complain of seeing colours, red, green, yellow, blue, black, etc.[2]

When the detachment is caused by tumour, the defect in the field is much more sharply defined, whilst the central vision remains unaffected. If, as is often the case, the tumour be peripheral,

[1] *Vide* Chap. IV. [2] *Vide* Chap. V.

the subjcetive symptoms are: sharply defined peripheral limitation of the field with good central vision.

If the detachment occur in both eyes, it is rare to find any great degrec of symmetry, such as is shown in Fig. 3, which is a chart

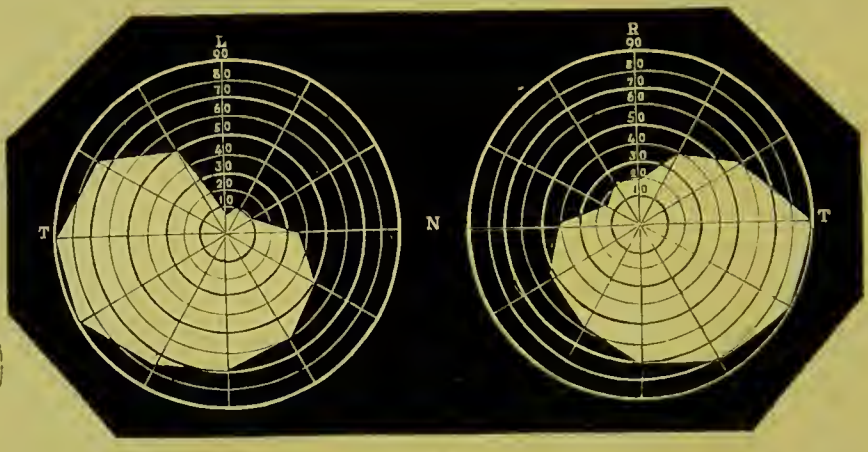


Fig. 3.

of the fields of H. H., a sailor, aged 28, after four months' duration of the disease, and in which V = $\frac{6}{200}$ and $\frac{20}{200}$.

Retinitis Pigmentosa.—The state of the peripheral vision in retinitis pigmentosa is often so characteristic as to render an ophthalmoscopic examination almost superfluous. The peculiar features are marked concentric limitation combined with relatively good central vision. Usually the objective appearances are also sufficiently striking to leave no doubt as to the nature of the case. In some instances, however, where there is an entire or almost entire absence of pigment, the subjective examination is of value in confirming the diagnosis. The extreme periphery of the retina does not appear to be subject in the same degree to the degenerative changes which constitute this disease, and we consequently find that, whilst the function of the greater part of the retina is lost, a zone or belt, or it may be only a small temporal portion, retains a part of its light. Fig. 4 represents the field of the left eye in a case which had existed from early childhood in probably pretty much the same condition. V $\frac{20}{40}$; M 3, 0. Curiously enough, this patient was completely colour-blind for red and green. There

can be little doubt that this was congenital, as in other cases I have invariably found the colour vision normal. If the ex-

Fig. 4.

ploration of the field in retinitis pigmentosa be merely undertaken with reference to its continuity and extent, whilst the relative activity in the functions of different parts is disregarded, it will be found that there is a great want of correspondence between the results arrived at at different times, unless great care be taken that the conditions are as nearly as possible similar at each examination. The state of the peripheral vision in retinitis pigmentosa is very markedly dependent on the intensity of light, a fact which is well known in connexion with the most prominent symptom of the disease, viz., night-blindness.[1]

Embolism.—A loss of half the field or a sector-shaped defect in it, *limited, however, to the one eye*, occurs in embolism of one of the principal branches of the central artery of the retina. The defect is generally in the upper or lower half of the field. This condi-

[1] *Vide* Chap. III.

tion is not infrequently called hemianopia (superior or inferior), although it is better to keep the term hemianopia for cases in which the half blindness is the result of a nervous lesion. After embolism of the main artery a small peripheral portion of the temporal side of the field retains often more or less completely its function, a fact which it is well to bear in mind, as the ophthalmoscopic appearances, after a certain time has elapsed, are often slight and undecided; and this condition occurring on the one side, especially in a subject the state of whose circulatory system would render embolism possible, is strongly suggestive of the blindness being due to this cause.

Scotomata occur either in connexion with lesions of the retinal elements, or, when independent of gross anatomical alterations, generally owing to some functional intra-cranial or cerebral disorder. The first may be of traumatic, inflammatory, or vasomotor origin, or be due to malformation. The cause is generally evident on ophthalmoscopic examination. The affected regions give rise to positive scotomata, of which the patient is most conscious when the centre is affected. This form is found in the different kinds of choroiditis, traumata, and hæmorrhages of the retina and choroid, coloboma, retinitis, etc. In diffuse retinitis a zonular or ring-shaped scotoma may often be found, either complete or interrupted at intervals, without any corresponding ophthalmoscopic changes. A form of choroiditis, which is limited to the region of the macula lutea, occurs in old people, and is often not very easily detected owing to the exudation, or senile changes of the basal membrane, differing only slightly in colour from the rest of the fundus. It gives rise to very different degrees of blindness, generally sufficient, however, to render the reading of newspaper type impossible. Owing to the limitation of the pathological change to the centre, complete blindness never results from it. Large or small insular scotomata not implicating the centre or extending to the periphery, for which no objective reason can be detected, are occasionally met with. The origin of these is obscure. Sometimes they appear to be the persistence in parts of the otherwise transient form of blindness characteristic of what is called scintillating amblyopia or am-

blyopia fugax.[1] The prognosis is good as far as blindness goes if the function of the surrounding portions of the retina remains normal, but the scotomata are not likely to disappear. Fig. 5 is the field of the left eye of a lady aged 40, in whom there were no ophthalmoscopic appearances or any history of exposure to strong light. The condition remained unaltered for several months, after which I did not see her again. The right eye was normal.

Glaucoma.—In this disease Von Græfe showed long ago that the condition of the field afforded an indication of primary diagnostic importance. The limitation which takes place is usually most marked to the nasal side (inwards and downwards).[2]

From an examination of a hundred cases exhibiting glaucomatous excavation, Bunge found the following forms of defect of the field of vision:—1. Defect of only the nasal portion of the field in 27 cases. 2. Defect predominating in nasal portion in 44 cases. 3. Field remaining forming a peripapillary oval in four cases. 4. Destruction of whole field including centre, with exception of temporal portion extending from blind spot to about 50° from centre in nine cases. 5. A central or paracentral scotoma with or without slight restriction of nasal periphery in four cases. 6. Restriction only upwards in two cases. 7. Concentric restriction in six cases. 8. Preponderance of defect in temporal half of field in four cases. The 88 cases under 1 to 5 inclusive, Bunge considers typical, the remaining atypical; thus he concludes as follows:—"For those forms of excavation of the optic nerve which on account of their form on the one hand, and on the other hand owing to many objective and subjective symptoms, such as increased tension, mydriasis, coloured vision, pain, etc., which occur at the same time, are recognised as glaucomatous excavations, that form of restriction of the field of vision is characteristic and typical which, beginning in the nasal periphery, afterwards affects the temporal periphery, but which, while slowly spreading in that direction, rapidly destroys the nasal half of the field, and gives rise to an

[1] *Vide* Chap. V.

[2] On this point Mauthner holds a different view. See his recent work on glaucoma.

oval field which stretches temporally from the macula to about 50°. Subsequently the functions of the central portions up to the nerve become also destroyed, and there remains vision only in a small temporal strip, which after wavering between existence and non-existence is also finally lost." Bunge considers that this typical series of successive invasion of definite portions of the field is

Fig. 5.

strongly suggestive of the excavation of the nerve being the cause of the limitation. The defects produced by choroiditis or optic atrophy are essentially different, while pressure on the nerves or vessels of the retina is an unlikely cause, as on such an assumption it would be difficult to explain why there is so frequently such a marked temporal sector of the field left relatively undisturbed. While there is much to be said in favour of this view, it must still be considered doubtful what circumstance it is which mainly leads to the defects of the peripheral vision in glaucoma.

It is interesting to observe the effect of iridectomy on the glaucoma field of vision. If the disease has been acute, the operation is generally followed by widening, sometimes to a very considerable

extent. This is also in some degree the case in chronic cases when the measurement of the field before operation has been made at a time when there has been considerable increase of tension, under which condition part of the limitation may be due more to a temporary abolition than to an irretrievable destruction of the local functional activity. In most chronic cases, however, and always in the true glaucoma simplex, there is cause for congratulation if the extent and acuity of the eccentric vision remains unaltered. Often the restriction becomes slightly greater, a result generally unimportant when compared to the chance of arresting the progress of destruction, but which occasionally—viz., when the restriction has at one part been close up to the point of fixation—may, by involving the centre, make all the difference between tolerably useful sight and an amount which is barely sufficient to enable the patient to move about without assistance.

According to Treitel, the boundaries of the eccentric colour vision in glaucoma are restricted only to the same relative extent as those for black and white. Undoubtedly such is often the case, and, as will be seen further on, this is entirely different from the condition in optic atrophy, so that if it be constant it would obviously afford an indication of some diagnostic importance in that class of cases in which the diagnosis between glaucoma simplex and atrophy is not without difficulty.[1]

Hemianopia.[2]—Symmetrical defects in the fields of vision are most commonly caused by lesions in the optic nerves, tracts or chiasma. If the defects lie to the same side in both eyes—that is, inwards in one and outwards in the other—the condition is that which is now generally called *homonymous hemianopia.* The hemianopia may be to the right or left (hem. homon. dextra v. sinistra), and partial or complete, just as we have complete and partial right or left hemiplegia. It may or may not be associated with hemiplegia. In the cases I have seen it has more often

[1] See also page 77.

[2] The term hemianopia is now adopted by most writers, and is obviously more consistent with the general terminology of nervous affections than hemiopia, which was formerly used to signify the same condition.

existed alone, but it is probable that those in which the visual paralysis forms but a part of the whole lesion will be more frequently met with in general hospitals. Of 29 cases observed by Sehweigger,[1] hemiplegia was absent in 16 and present in 13, though in most of these it was only slight and transitory. If the case be recent and uncomplicated, there is no appearance of atrophy of the nerves. The line of demarcation between the blind and seeing portions of the field is usually sharp and regular; when the hemianopia is complete it is a line coinciding (for the peripheral portions of the field, at any rate) nearly, if not exactly, with the vertical through the point of fixation. Whether or not it also

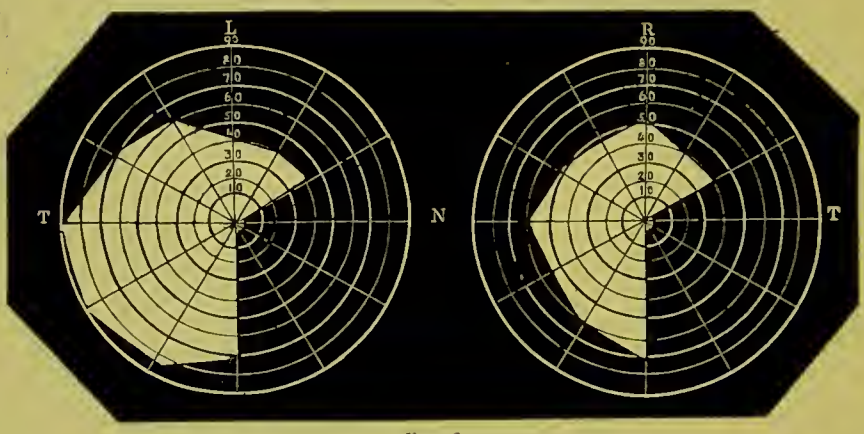


Fig. 6.

passes vertically through this point, indicating an interruption in the functional activity of exactly half the macula lutea, is still a disputed point. My own examinations of cases of homonymous hemianopia lead me to believe that in some cases it does so, and in others not. I have most frequently found the immediate neighbourhood of the point of fixation (from 2°–5°) apparently not implicated, but this may possibly be owing to the difficulty of securing accurate fixation, as some have suggested.[2] The lesion producing homonymous hemianopia has been found to occupy

[1] Graefe's *Archiv*, xxii. part 3.

[2] On this point see Mauthner, *Hirn und Auge*, No. 5 of his excellent series of lectures on ophthalmological subjects.

different situations, viz., the optic tract, basal ganglia fibres of Gratiolet, and the cortex of the brain in the occipital lobe.

The experiments of Munk, and the results of post-mortem examination in the cases of Hoseh, Curschmann, Haab, Samelsohn, and a few others less absolutely conclusive, have definitely established the existence of a unilateral source of innervation for corresponding halves of both retinæ. In the cases where the lesion has been cortical, it has either been one occupying a

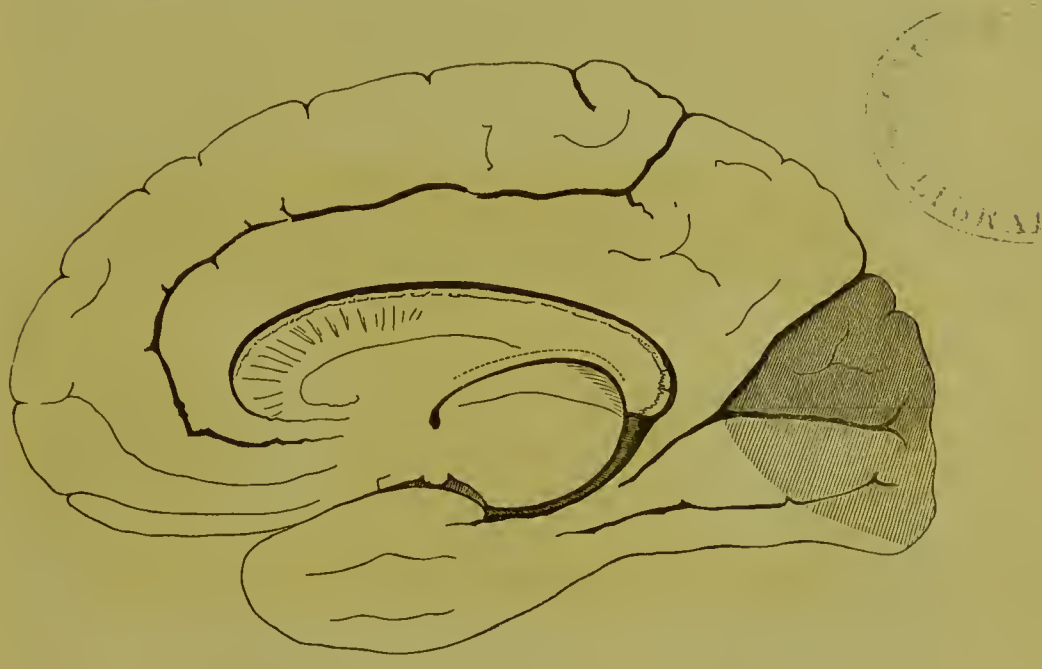


Fig. 7.

portion of the grey matter of occipital lobe, or pressing on the white matter with which it is in connexion, so that there is little doubt now that Ferrier's experiments which led him to localize the visual centres in the angular gyri, have given rise to the hemianopic symptoms owing to a wounding of the fibres of Gratiolet. The shaded part in Fig. 7 represents the portions in which lesions giving rise to homonymous hemianopia have most frequently been found to be located. The denser shading covers the presumable centre (inner portion) of vision for the left halves of the fields of vision. Important data in connexion with the localizing of lesions which have led to hemianopia are afforded by the

consideration of the concomitant symptoms as well as by the extent and configuration of the blind area. A very able attempt has recently been made by Wilbrand[1] to make use of the data afforded by the thorough subjective examination of cases of hemianopia, in order to arrive at a refinement in the diagnosis of the site of the lesion. Wilbrand's work, though very suggestive, is necessarily to a considerable extent theoretical. He keeps constantly before him, however, the results of numerous post-mortem examinations which he has collected, and the views he expresses are not inconsistent with the known pathology of the subject. An analysis of the subjective symptoms of hemianopia cases shows that:—1. When in any homonymous portions of the fields of vision there is a defect in the light sense, the defects in the senses of form and colour are at least quite as extensive. 2. When the light sense remains intact, but a homonymous defect exists for form, this is always accompanied by at least as extensive a defect for colours. Wilbrand therefore concludes that the nerve fibres connected with the centres for the senses of colour and form pass through the light sense centre. As, too, there are cases on record where the colour sense alone in the halves, or in some particular homonymous sections of the fields of vision, had disappeared, as well as cases where both colour sense and form sense had disappeared from similar portions, leaving, however, the light sense intact in these portions, Wilbrand draws the further conclusions that the centre for form must lie between those for colour and light, while that for colour must at the same time occupy the most central position in the brain. Many examples of actual cases are given in Wilbrand's monograph of all the combinations which are possible on the above assumptions.

When the hemianopia is partial, the defect is generally, though not always, of equal extent in both eyes. Figs. 8 and 9 are examples of both forms which I have met with.

Hemianopia remains, as a rule, stationary, even although the subjective symptoms become in many cases, after some time,

[1] *Ophthalmiatrische Beitraege zur Diagnostik der Gehirn-Krankheiten.* (Wiesbaden, 1884.)

associated with more or less visible changes in the discs. According to Mauthner, the most marked atrophic discoloration is found in the eye of the side to which the field is restricted. This point requires, however, further confirmation.

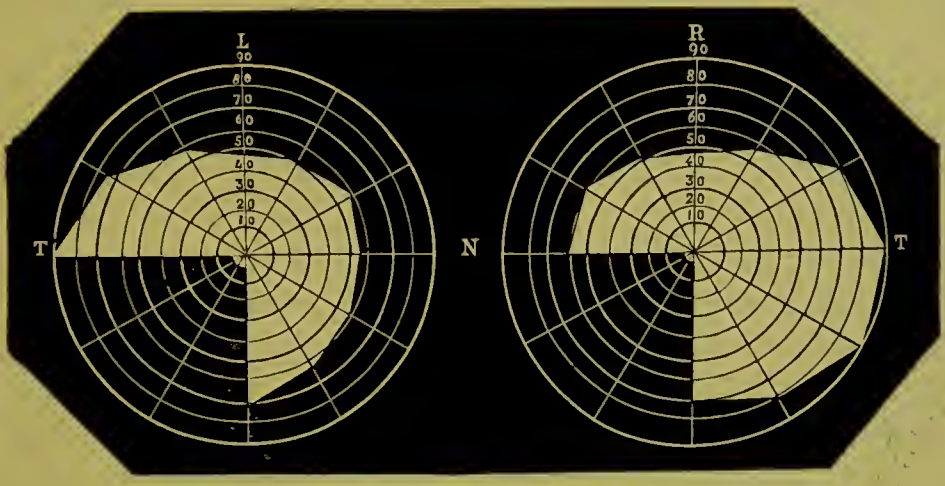


Fig 8.

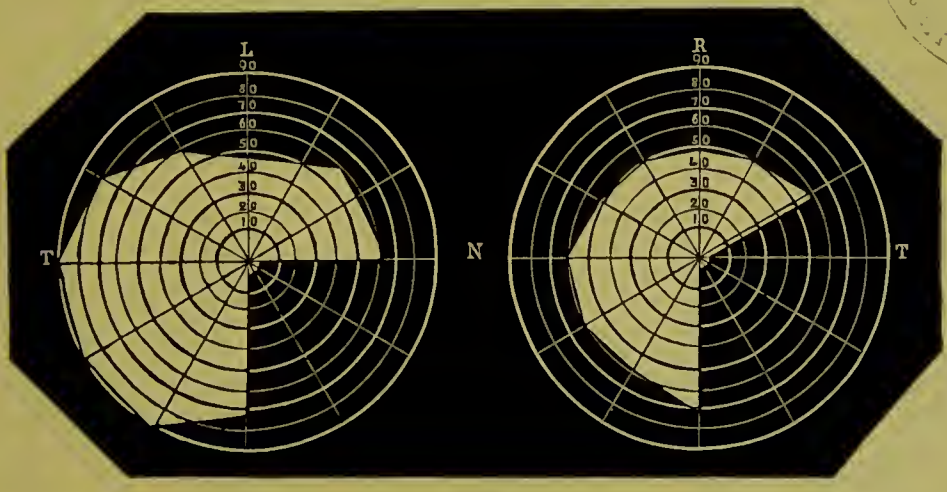


Fig. 9.

A very unusual form of hemianopia is described by Foerster in his chapter on the relation of ocular and visual disorders to general disease, which forms one of the most interesting and novel features of the well-known Græfe-Sæmisch *Handbuch* (vol. vii. chap. 13). In this form the defect is limited to corresponding insular portions in the homonymous halves of the two visual fields.

Two other forms of hemianopia occur, viz., temporal and nasal

hemianopia. In the former the outer, and in the latter the inner, half of each field of vision is deficient. They are both much rarer than the homonymous form.

The arrangement of the optic nerve fibres renders the localization of the lesion producing temporal hemianopia a very simple process. There is, of course, the remote possibility of a symmetrical lesion in the two halves of the brain, but apart from this, and as by far the most probable cause of the symptoms, we must admit an interruption of function produced by some destruction or compression of the nervous elements of a portion of the chiasma. The defects produced in the function of the temporal halves of each retina will be the more complete and symmetrical the more the lesion is confined in its effect to an antero-posterior line through the chiasma. Thus, whereas the lesion producing the more common form of homonymous hemianopia may be anywhere along the course of the optic nerve fibres between the chiasma and the cortex of the occipital lobe, and can rarely be localized with any great certainty, although concomitant symptoms often afford a clue, temporal hemianopia is almost certainly due to a lesion mainly implicating the chiasma.

It is difficult to form any idea of the frequency of this form of hemianopia. Owing to the nature of the defect leaving a much larger field for both eyes than the homonymous form, the patient does not, as a rule, complain of blindness to one side. The other symptoms too, amblyopia, ophthalmoscopic changes, etc., are sufficiently marked to engage the whole attention of anyone who does not make a practice of examining the peripheral vision. Besides, it is not likely that all the cases observed are published. Mauthner believes that they constitute about 1 per cent. of all cases of hemianopia, while Foerster's estimate is 23 per cent. The actual percentage probably lies between these extremes and much nearer the former than the latter. It is obviously a disease of too rare occurrence to furnish even approximately correct statistics from the practice of any single observer, however extensive his experience. Græfe in his famous paper on the examination of the field of vision in amblyopic affec-

tions,[1] which first directed attention to the importance of determining accurately the state of peripheral vision, says that whereas one not unfrequently meets with cases in which the vision is lost from the same side of each eye, it is not by any means a frequent occurrence (es nicht gar häufig vorkommt) to find the right half of the right and the left half of the left eye defective.

The literature of the subject has been very fully collected by Mauthner[2] and Wilbrand.[3] The salient points in connexion with 37 cases which had been recorded up to the date of the publication of their works are given by these authors.[4] Several of these cases cannot, however, be looked upon as undoubted examples of this disease (looked upon as a manifestation of central changes). Since these works were written several other cases have been published.

Five cases have come under my own observation. Although I have elsewhere[5] published an account of the two first, the comparative rarity of this form of half-blindness is perhaps a sufficient excuse for referring to them here, more especially as they illustrate well two forms in which this defect manifests itself.

Case I.—John B., cork cutter, aged 28, first seen 28th March 1882, when he stated that his sight had been failing for some months. On ophthalmoscopic examination, the discs were found to be extremely pale. V = $\frac{20}{100}$ L, $\frac{20}{40}$ R. The acuity of vision did not materially change within the limits of illumination by which I retained full vision. The vision of the temporal half of the field of the left eye was abolished (retaining only slight perception of light), and in the right eye the temporal side of the field had also suffered a very considerable impairment of its function (loss of colour vision and very defective form sense). The portions of the fields retained in both eyes seemed to be normal in every respect, and there was a

[1] *Archiv. f. Ophth.*, vol. ii. 2, p. 258.

[2] Hirn und Auge.

[3] Ueber Hemianopsie, und ihr Verhältniss, zur topischen Diagnose der Gehirnkrankheiten.

[4] Thirteen cases are given by both, 14 by Wilbrand alone, and 10 by Mauthner alone ; W. also quotes 12 cases of autopsy in disease of chiasma.

[5] *Ophthalmic Review*, 1884.

sharp line of demareation between the healthy and defective portions. This boundary was nearly vertieal, and apparently about 3°–4° to the temporal side of the middle line through the centre; the extent of the area of blindness being, however, greater in both eyes below than above. There was a doubtful syphilitie history beginning twelve years previously, whieh led me to diagnose a periostitis or gummatous swelling, exerting a pressure on the chiasma. The patient did not return for a year afterwards, at which time the condition appeared to be very much the same, with the exception that both temporal halves of the field were now blind. His own statements were to the effect that he had been mueh better for eight or nine months, and had then got worse again. A complete examination of the central and peripheral vision was not made then, as he promised to eome to the hospital next day. This he failed to do, however, and next made his appearance on the 29th of November 1883, when the eondition

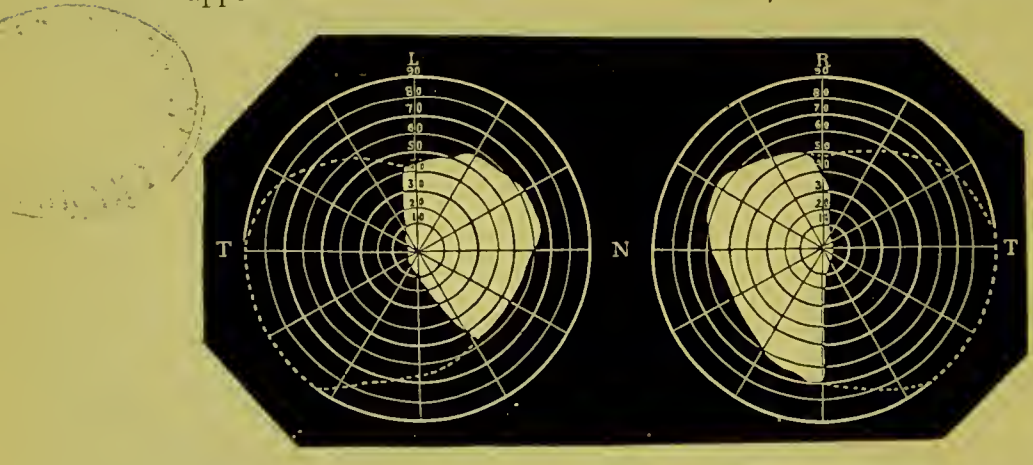


Fig. 10.

of the fields of vision was as in Fig. 10, with V $> \frac{20}{200}$ R, $> \frac{20}{70}$ L., only slightly worse, therefore, than eighteen months previously—ophthalmoscopieally; marked atrophy of both nerves. After this a rapid deterioration took place, and when he was admitted, on 8th February, as an in-patient under Dr Wyllie, the central vision was extremely defeetive, although the hemianopic nature of the blindness remained still marked. I had reeommended him to seek admission some time previously, but he declared that an

irresistible *drowsiness*, which lasted for days, had prevented him from coming. He was discharged on 23rd March almost quite blind, but otherwise apparently in better health; and the following was Dr Wyllie's report on 29th April:—Remains perfectly blind, except very faint perception of window; says, however, that on some days he sees objects pretty distinctly, being able, on such occasions, for example, to recognise his mother. This patient afterwards became insane, and was removed to the Asylum at Morningside, where he died in July 1886.

On post-mortem examination an aneurism ($2\frac{3}{4}'' \times 2\frac{1}{4}'' \times 1\frac{1}{2}''$) was found springing from the right internal carotid artery. The chiasma and nerves were so much disorganized by the aneurism that they were difficult to trace, and a considerable depression had been made in the sella turcica. The preparation is in the possession of Dr Spence. The occasional improvement which took place in this case before permanent blindness set in is suggestive, considering the nature of the lesion.

This is an instance of a case in which the typical temporal blindness was slowly developed, remained for a considerable time unaltered, and then proceeded to more complete blindness. The cases of del Monte,[1] Mooren (second case),[2] Hirschberg,[3] and others are more or less similar, and it seems probable that the prognosis is not so favourable in the slowly progressing cases as in such as the following, where there was a gradual but steady improvement from the first:—

Case II.—Mrs M'A., 32, from Dundee, first presented herself at the Royal Infirmary on 22nd August 1883, complaining of defective sight. On ophthalmoscopic examination both discs were found to be extremely pale; there was, besides, a very abnormal arrangement of the pigment cells in the choroidal interspaces, probably congenital, V almost nil L, $\frac{20}{100}$ R, and blindness of right side of field. A chart of the field was unfortunately not taken. She could

[1] *Osservazioni e note cliniche*, Naples, 1871.

[2] *Ophthalmiatrische Beobachtungen*, 1867.

[3] *Beiträge zur practischen Augenheilkunde*, 3.

give no cause for the blindness, but had been suffering for six months from headaches, constipation, giddiness, and flushing of the head. Menstruation ceased at the age of 23, and there were no children. I recommended a course of saline waters and cold douches, which she adopted; and when she next returned, on 18th October, there was very considerable improvement in vision: L = fingers at several feet, R = $\frac{20}{70}$. The state of peripheral vision at that date is shown by the chart, Fig. 11. The blindness of the left

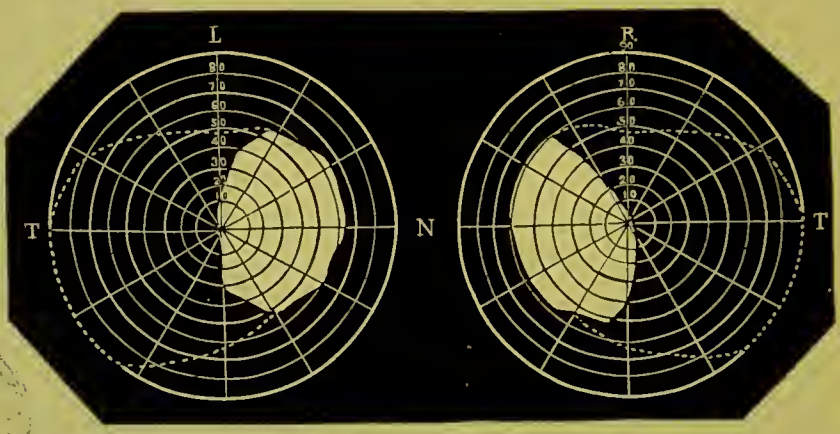


Fig. 11.

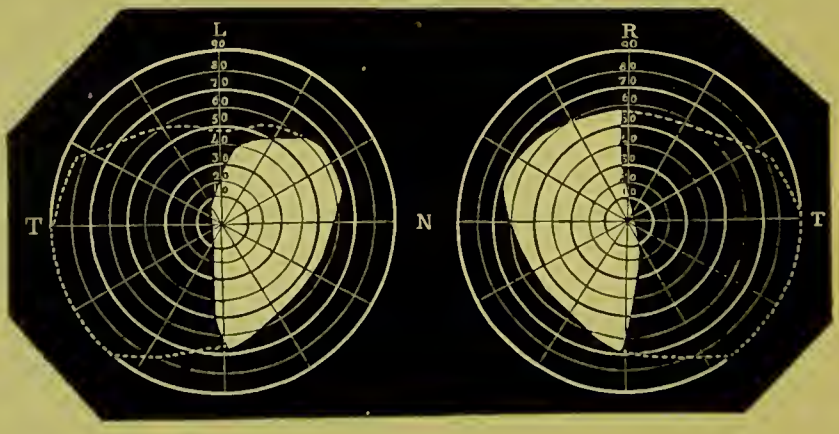


Fig. 12.

eye at the previous visit had obscured the real nature of the case. Shortly after this she seems to have had some degree of polyuria, lasting for two months (there were frequent calls for micturition,

and a larger quantity of urine passed each time than usual). On 19th April 1884 the patient presented a much more healthy aspect, the headaches and giddiness, etc., had disappeared, and the vision had risen to $\frac{20}{200}$ L and $\frac{20}{50}$ R, with field as in Fig. 12.

The lesion in this case had evidently not been a spreading one, and must have produced the maximum destruction or pressure antero-posteriorly along the chiasma, probably a hæmorrhage or embolism (?). The cases of Laqueur,[1] Græfe,[2] and Schön[3] (second case), and others, are analogous in the manner of their development and course, Græfe's ending in complete cure.

Polyuria is mentioned as a complication in four cases—those of Græfe, del Monte, Brecht,[4] and Nieden. In 153 of homonymous hemianopia collected by Wilbrand, it was met with but twice.

It seems possible that the cessation of menstruation may have been of some etiological importance in the case of Mrs M'A. A similar circumstance occurred in two of Schön's cases, in one of which menstruation ceased at the age of 24, in the other at 36. In Uhthoff's[5] case the catamenia did not return after a confinement in the 27th year, and in the case already cited of Græfe's, the hemianopia occurred after cessation during two periods. All these cases suffered much from headache and giddiness, and although the diagnosis arrived at by V. Græfe in his case was limited periostitis, the case could as well be explained on the assumption of some vascular change.

Case III.—Wm. C., aged 23, only came under my notice a week before death, in August 1885. The vision was then greatly impaired in both eyes, but there was a very marked temporal hemianopia, the temporal halves of each field being absolutely blind. On post-mortem examination a tumour the size of a small hen's egg was found occupying the region of the pituitary body, and involving apparently completely the chiasma. The preparation is in the possession of Dr Byrom

[1] *Klin. Monatsblätter*, 1864, p. 276.
[2] *Klin. Monats.*, 1865.
[3] *Die Lehre vom Gesichtsfelde, etc.*, 1874.
[4] *Græfe-Sæmisch*, v., 938.
[5] Græfe's *Arehiv*, xxvi. 1, p. 263.

Bramwell. Unfortunately no microscopic examination of the fibres of the chiasma could be made, as decomposition was too far advanced before it came into his hands, so that he was unable to determine the area of greatest destruction.

Case IV.—H. B., aged 40, first seen on 4th December 1885, although the affection had apparently already existed for about two years. No history could be given as to how it began. There had certainly been no syphilis. At the first examination V = > $\frac{20}{200}$ left, $\frac{20}{40}$ right. There was complete loss of each temporal portion of the field, with a nearly vertical line of demarcation in each eye, passing, however, rather further in, in the case of the left than of the right. I recommended absolute rest and change for three months. In the meantime he consulted Dr Byrom Bramwell, Dr Emrys Jones, Dr Hughlings Jackson, and others, and on 1st April 1886, when I next saw the patient, the condition was exactly the same as on the former examination. Since then there has been an undoubted deterioration. The treatment which has been adopted by those whom he has consulted has been mainly the internal use of iodide and bromide of potassium, and mercurial preparations. These have certainly done no good. The optic discs are now very pale. He has headaches, but no vomiting The diagnosis appears therefore uncertain, although I had at first classed this case in the stationary and relatively harmless group.

Case V.—W. W., aged 53, has only recently come under my observation. The blindness for which he consulted me came on with headaches, and rapidly, so that the condition after a week's duration was as follows:—V = $\frac{20}{200}$ R, $\frac{16}{200}$ L. Both temporal halves of the fields of vision lost (or rather extremely defective) up to about 5° from the points of fixation. The circulation was extremely slow, and Dr Byrom Bramwell, who saw him at this time, considered him to be suffering from an acute increase of intracranial pressure. There was, however, no neuritis. The condition after one week had immensely improved. V = $\frac{20}{20}$ R., and $\frac{20}{30}$ L; while about $\frac{2}{3}$ of each temporal field had more or

less regained its functional activity. In this case the symptoms were evidently purely the result of pressure on the chiasma.

Nasal hemianopia is of such rare occurrence that it hardly deserves attention. It appears to be due to symmetrical lesions of the optic tracts, and has probably in most cases only been the beginning of complete optic atrophy.

Optic Atrophy.—Except where there is marked diminution in the size of the retinal arteries, it is only when the pallor of the discs is extreme that we are justified in diagnosing the existence of optic atrophy from ophthalmoscopic examination alone, as the physiological limits of colour and excavation are extremely wide. Along with a low degree of central visual acuity, an excessive pallor of the nerve is certainly suspicious; but there are many circumstances under which those two conditions exist, either mutually dependent or not, unconnected altogether with atrophy,—for instance, all those which give rise to a central negative scotoma. For a safe diagnostic as well as prognostic criterion we are thrown back upon the determination of the state of the field of vision. Our knowledge on this subject was first clearly formulated by V. Græfe,[1] and, indeed, so thoroughly was this subject investigated by him, that, with the exception of the slight refinement which has been introduced by the examination of the peripheral colour vision, little or no advance has since been made. One of the most important points which resulted from his observations was, that however much the central vision might be diminished, no fear of progressive atrophy, and therefore eventual total blindness, need be entertained if the boundaries of the field of vision remain normal. In progressive atrophy there is *invariably* a limitation of the field. The limitation often takes place all round (concentric limitation), but in other cases the field in one direction is more restricted than in the rest. The restriction which is taking place can frequently be most easily demonstrated for colours. This is, very probably, simply because, if the examination be made in ordinary daylight, the corresponding failure of the sense of form may, if slight, more readily escape detection. There does not appear to be any reason

[1] *Klinische Monatsbl. f. Augenh.*, 1865, and *Archiv*, vol. ii.

for supposing that the failure of the colour perception is unconnected with that of the other functions of the visual apparatus, though, considering the number of possible seats as well as natures of the lesions which would disturb the functions of the optic nerve, the possibility of such an independence should be borne in mind. Before, however, we are able to form correct conclusions on the nature of the colour failure in cases of optic atrophy, it would be necessary to have more ample data of the normal relations connecting the three senses of light, form, and colour. It is certain that they are all different functions of the special sense of vision, but in how far mutually dependent remains to be shown. Although it is most common to find, in cases of optic atrophy, that the failure of the functions of the central and peripheral portions of the retina advance hand in hand, still cases are met with in which the peripheral functions have suffered much more severely than the central. The long time which sometimes elapses between the beginning of atrophy and complete blindness renders it difficult to give a favourable prognosis based on an apparent arrest in the progress of the amblyopia; but if the condition of the field of vision remain absolutely stationary for several months, the prognosis is certainly hopeful, and all the more so if the cause of the atrophy appears to be at an end. Cases occur in which, after the disease has produced a considerable amount of amblyopia along with great limitation of the field, it ceases, and the patient retains what is left. Such cases are generally ones in which the atrophy is secondary to neuritis.

It is impossible from the functional examination to determine whether the atrophy is connected with spinal or cerebral disease, or due to some other cause (hæmorrhage, primary sclerosis of the optic nerve, etc.), but there are other circumstances which render one or other cause the most probable in any case. In a great many of the cases of spinal atrophy some ataxic symptoms are already present when the amblyopia begins, whereas it is otherwise with the atrophy of cerebral origin, which is seldom preceded, though often followed, by mental disturbances. In cases of atrophy there is generally a considerable difference in the

amblyopia of the two eyes. It is not uncommon to find the one far advanced towards amaurosis whilst the vision is still very fair in the other. The mode of development of the amblyopia is, nevertheless, generally much the same in both. Thus, if any particular portion of the field is most restricted in the one, the first symptoms may be looked for in the corresponding part of the field of the other eye.

The characteristic subjective symptoms of optic atrophy are, then, more or less concentric limitation of the field of vision, which is greatly more pronounced for colour, with a simultaneous diminution of the central visual acuity. There is no constant relation between the amount of failure of peripheral and central visions, although a large proportion of cases present much the same conditions.

In some cases the central vision remains for a long period tolerably normal, although the restriction of the field is extreme. Such small fields with good central vision are, as we have seen, more characteristic of retinitis pigmentosa, but the possibility of their occurring in progressive atrophy should be remembered. I have met with a case in which the fields of vision were both so restricted that there appeared to be nothing left but central vision, which was normal, and to account for which there were no abnormal ophthalmoscopic appearances. I am indebted to Dr Charnley for notes of a somewhat similar case from Moorfields Hospital. Such cases appear to belong to another category from progressive atrophy. Possibly they may be cases of double homonymous hemianopia, caused by lesions which do not involve the centres for the macular fibres. More probably they are a variety of reflex amblyopia.

Central negative scotomata are met with in a class of cases which are almost always, though probably not exclusively, of toxic origin. The scotoma, which may be so slight as only to be readily demonstrated for colours, takes the form of a horizontal oval, stretching from the portion of the field corresponding to the position of entrance of the optic nerve to slightly beyond the point of fixation. The condition is accompanied by a diminution

of central visual acuity of very varying amount. In by far the greatest number of cases—and in such the prognosis is extremely

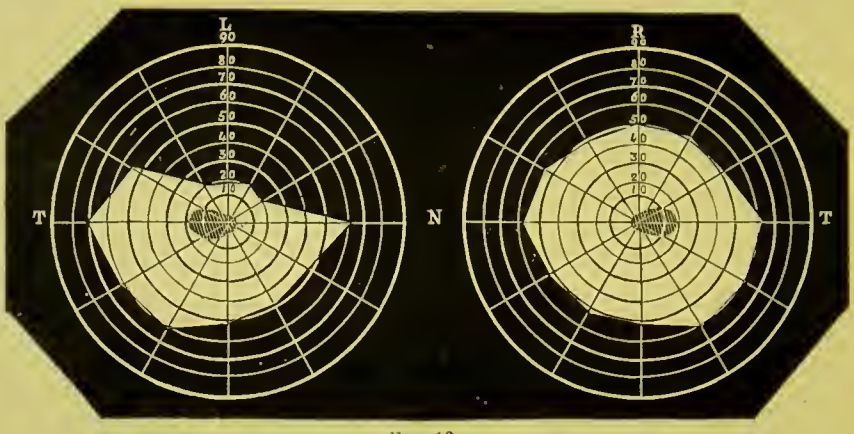


Fig. 13.

favourable if the cause of the amblyopia be removed—no other defect of the field of vision exists, but a typical central negative scotoma may be complicated in rare cases by other limitations. In these it is doubtful whether complete recovery is ever possible, and, indeed, it is not impossible that they may ultimately lead to complete blindness. Fig. 13 gives the field in a case of the complicated form.

The vision of the right eye in this case improved during treatment from $\frac{15}{200}$ to $\frac{10}{100}$; that of the left remained stationary at $\frac{8}{200}$. The ophthalmoscopic changes were slight.

Most frequently this condition is the result of tobacco poisoning. The toxic effects of the tobacco get the upper hand, when owing to chronic alcoholism, or any other debilitating influence, the nervous system is unable to withstand it. In a large proportion of cases smoking has been freely indulged in at times, when the counter stimulus of food has been absent, as in the morning before breakfast, or during the night. The fibres whose function is diminished in central or toxic amblyopia have been lately found to constitute a distinct bundle, occupying something like one-quarter of the whole optic nerve. They have been called the papillo-macular fibres, and although recent investigations point to a neuritis of these fibres, as the lesion producing the characteristic

central scotoma, it would seem extremely improbable, owing to the complete curability of the large majority of cases in which tobacco is the prime cause of the symptoms, that such an inflammation is at all frequent.

From the preceding short sketch of the state of peripheral vision in different diseases it is evident that there are many points of diagnostic importance to be gathered from an examination of the field. This, in the case of the diagnosis between intra-ocular and extra-ocular cause, which, so far as our present knowledge goes, is the direction in which the observed differences are most pronounced, is not so necessary, as there are, as a rule, objective signs which lead us to the same conclusions. But there can be little doubt that in the thorough examination of the relative as well as absolute functional activity of all parts of the retina we possess a means of immense value in the localization of intra-cranial disease. But it is not merely as a method of diagnosis that this examination deserves attention; in many cases it affords the most delicate means of ascertaining the course taken by any disease, especially when of intra-cranial origin. Here, however, it is necessary to bear in mind the circumstances which have been already mentioned as influencing the result of the examination, viz., intensity of light, practice, etc., so as not to ascribe, without good cause, slight or sometimes even considerable alterations in these results to progress of the disease either in the direction of recovery or blindness.

The prognostic importance attaching to the state of the field has been alluded to under the different affections, but it may be well to recall a few points here. The prognosis is good if there be diminution of central vision without any limitation of the field, or if the limitation takes the form of homonymous hemianopia in which the portions affected are *sharply* separated from those in which the function is retained. If, on the other hand, there be limitation of the field, even without any apparent cause, the probability of subsequent atrophy and blindness is very great, and this is all but certain if there be a previous atrophy of one eye. A good deal of prognostic significance, however, attaches to the

time which elapses for the production of a defect in the field. Generally speaking, the limitations which come on suddenly are not so liable to end in atrophy as those of slow, gradual invasion; but here again we must keep in view the known or probable nature of the central cause, as it is evident, for example, that any sudden alteration appearing after a lesion of traumatic, apoplectic, or even embolic origin would necessitate a more serious prognosis than a similar change owing its existence probably to vasomotor, hysterical, reflex, or other more or less obscure functional disorders.